PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME

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DECLARATION

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To

My Family and

Respected

Teachers

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Dr. Anchal Aggarwal

ABBREVIATIONS

LGBS	Landry Guillain Barre Syndrome
GBS	Guillain Barre Syndrome
AIDP	Acute Inflammatory Demyelinating polyradiculoneuropathy
MFS	Miller Fisher Syndrome
AMAN	Acute Motor Axonal Neuropathy
AMSAN	Acute Sensorimotor Axonal Neuropathy
US	United States
SIADH	Syndrome of Inappropriate Anti- diuretic Hormone Secretion
CIDP	Chronic Inflammatory Demyelinating Polyradiculoneuropathy
CSF	Cerebrospinal Fluid
NCS	Nerve Conduction Studies
EMG	Electromyography
ICU	Intensive Care Unit
NIF	Negative Inspiratory Force
IVIg	Intravenous Immuneglobulin
VC	Vital Capacity
TLC	Total Lung Capacity
RV	Residual Volume
NPV	Negative Predictive Value
FVC	Forced Vital Capacity
CMAP	Compound Muscle Action Potential
p/d CMAP	Proximal to Distal Compound Muscle Action Potential ratio
MRC	Medical Research Council
NCV	Nerve Conduction Velocity
LLN	Lower Limit of Normal
DMCH	Dayanand Medical College and Hospital

SBC	Single Breath Count
FCR	Flexor carpi Radialis
DIP	Distal Interphalangeal
FCU	Flexor Carpi Ulnaris
SCM	Sternocleidomastoid
SD	Standard Deviation
SPSS	Statistical Package for the Social Science
OPD	Outpatient Department
UL	Upper Limb
LL	Lower Limb
CPN	Common Peroneal Nerve

ABSTRACT

Introduction: Landry Guillain Barre Syndrome is an acute fulminant polyradiculoneuropathy occurring annually at a rate of 1-4 cases per 1,00,000. It is one of the most common causes of neuromuscular respiratory failure with 20-30% patients requiring mechanical ventilation as respiratory failure contributes to significant morbidity and mortality in these patients.

Aim of Study: To study various phrenic nerve conduction abnormalities and to see if they can predict impending respiratory failure in LGBS patients.

Materials & Methods: This was a prospective study conducted on patients of age ≥18 years who presented to DMCH Ludhiana from Nov, 2018 to Apr, 2020 with rapidly evolving areflexic ascending paralysis or quadriparesis or having the suspicion of having LGBS and NCV studies including phrenic nerve were done and patients were followed up for development of any respiratory abnormalities during hospital stay. Various clinical and electrophysiological parameters were then co-related if they predicted respiratory involvement in LGBS patients.

Results: Total 47 patients were enrolled and 29.8% (14 patients) of them had respiratory involvement with 64.3% of these (9 patients) requiring mechanical ventilation**.** Age and gender had no co-relation with respiratory involvement. Mean SBC was 16.38 ± 7.35 in patients who progressed to respiratory involvement. Power grade ≤ 3 suggested severe disease and more chances of progression to respiratory failure. Phrenic nerve conduction studies showed decreased amplitude or increased latency or both or no response at all but these findings alone were not predictive of respiratory involvement in LGBS patients. Peripheral motor nerve studies might help in predicting respiratory involvement and needs further evaluation with larger sample size.

Conclusion: In our study Phrenic nerve conduction abnormalities observed were decreased amplitude, increased latency or both or no response at all. Phrenic nerve conduction studies alone cannot predict respiratory involvement in LGBS patients.

Keywords: Landry Guillain Barre Syndrome, NCV, Phrenic Nerve, Respiratory involvement.

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-7
II	AIMS AND OBJECTIVES	8
III	REVIEW OF LITERATURE	9-14
IV	MATERIALS AND METHODS	15-21
V	OBSERVATIONS AND RESULTS	22-60
VI	DISCUSSION	61-72
VII	SUMMARY AND CONCLUSION	73
VIII	BIBLIOGRAPHY	74-82
	APPENDICES – I - PROFORMA - MASTER CHART	
	APPENDICES – II - APPROVAL LETTER OF THESIS PLAN - THESIS PLAN	

LIST OF TABLES

TABLE NO.	TITLE	PAGE NO.
1.	Electrodes placement	17
2.	Normal values	18
3.	Respiratory involvement seen	22
4.	Patients who required mechanical ventilation	23
5.	Patients with respiratory involvement progressing to mechanical ventilation	24
6.	Age distribution	25
7.	Age differences in both groups with and without respiratory involvement	26
8.	Influence of age on respiratory involvement in LGBS	26
9.	Gender distribution	27
10.	Gender distribution and influence on respiratory involvement in LGBS	28
11.	Respiratory rate on admission predicting respiratory deterioration in LGBS	29
12.	Mean SBC in LGBS	30
13.	Power grading and co-relation with respiratory involvement in LGBS	32- 33
14.	Phrenic nerve motor amplitude on left side	35
15.	Co-relation of left phrenic nerve motor amplitude with respiratory involvement	36
16.	Phrenic nerve motor amplitude on right side	37
17.	Co-relation of right phrenic nerve motor amplitude with respiratory involvement	38
18.	Bilateral phrenic nerve amplitude involvement	39
19.	Bilateral phrenic nerve motor amplitude involvement with respiratory involvement	40
20.	Phrenic nerve motor latency on right side	41

21.	Co-relation of right phrenic nerve motor latency with respiratory involvement	41
22.	Phrenic nerve motor latency on left side	43
23.	Co-relation of left phrenic nerve motor latency with respiratory involvement	43
24.	Bilateral phrenic nerve latency involvement	45
25.	Bilateral phrenic nerve motor latency with respiratory involvement in LGBS	45
26.	Bilateral phrenic nerve amplitude as well as latency involvement	47
27.	Bilateral phrenic nerve amplitude as well as latency involvement predicting respiratory involvement in LGBS	47
28.	Peripheral motor nerves' distal latency co-relation with respiratory involvement in LGBS	50
29.	Peripheral motor nerves' amplitude co-relation with respiratory involvement in LGBS	53
30.	Peripheral motor nerves' velocity co-relation with respiratory involvement in LGBS	56
31.	LGBS variety and relation with respiratory involvement	58
32.	Cranial nerve involvement in LGBS	59
33.	Cranial nerve involvement role in predicting respiratory involvement in LGBS	60

LIST OF FIGURES

FIGURE NO.	TITLE	PAGE NO.
1.	Respiratory involvement seen	23
2.	Patients with severe disease requiring mechanical ventilation	23
3.	Patients with respiratory involvement progressing to mechanical ventilation	24
4.	Age distribution	25
5.	Respiratory involvement according to age in LGBS	26
6.	Gender distribution	27
7.	Influence of gender on respiratory involvement in LGBS patients	28
8.	On admission mean respiratory rate more in those progressing to respiratory involvement	29
9.	Mean SBC lesser in patients with respiratory involvement	30
10.	Power grading predicting respiratory involvement in LGBS in upper limbs	34
11.	Power grading predicting respiratory involvement in LGBS in lower limbs	34
12.	Left phrenic nerve motor amplitude findings	35
13.	Left phrenic nerve motor amplitude relation with respiratory involvement in LGBS patients	36
14.	Right phrenic nerve motor amplitude findings	37
15.	Right phrenic nerve motor amplitude relation with respiratory involvement in LGBS patients	38
16.	Bilateral phrenic nerve amplitude abnormality	39
17.	Bilateral phrenic nerve amplitude involvement relation with respiratory involvement in LGBS patients	40
18.	Right phrenic nerve latency findings	41

19.	Right phrenic nerve motor latency relation with respiratory involvement in LGBS patients	42
20.	Left phrenic nerve motor latency findings	43
21.	Left phrenic nerve motor latency relation with respiratory involvement in LGBS patients	44
22.	Bilateral phrenic nerve latency abnormality	45
23.	Bilateral phrenic nerve motor latency involvement relation with respiratory involvement in LGBS patients	46
24.	Bilateral phrenic nerve amplitude and latency involvement	47
25.	Bilateral phrenic nerve motor amplitude and latency involvement relation with respiratory involvement in LGBS patients	48
26.	Relation of peripheral motor nerve latency findings with respiratory involvement in LGBS patients	51
27.	Relation of peripheral motor nerve amplitude findings with respiratory involvement in LGBS patients	54
28.	Relation of peripheral motor nerve velocity findings with respiratory involvement in LGBS patients	57
29.	Frequency of LGBS abnormality types	58
30.	Type of LGBS variety and relation with respiratory involvement	59
31.	Cranial nerve involvement in LGBS	60
32.	Relation of cranial nerve involvement with respiratory involvement in LGBS patients	60

INTRODUCTION

Landry Guillain Barre Syndrome (LGBS) is an acute immune-mediated polyneuropathy, usually provoked by a preceding infection, which occurs worldwide and affects all age groups. Initially, Guillain Barre Syndrome (GBS) was considered a single disorder but now is recognized as a heterogeneous syndrome with several variant forms like acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the Miller Fisher syndrome (MFS), acute motor axonal neuropathy (AMAN) and acute sensorimotor axonal neuropathy (AMSAN).

An antecedent infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of sharing of cross-reactive epitopes, known as molecular mimicry[1] resulting in acute polyneuropathy. This immune response can be directed towards the myelin or the axon of the peripheral nerve. Immune reactions directed against epitopes in myelin can cause acute inflammatory demyelinating neuropathy (AIDP) and those against epitopes contained in the axonal membrane cause the acute axonal forms of GBS: acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) [2].

GBS occurs world-wide with an overall incidence of 1 to 2 cases per 100,000 per year [1, 3]. While all age groups are affected, the incidence increases by approximately 20 percent with every 10-year increase in age beyond the first decade of life. In addition, the incidence is slightly greater in males as compared to females.

The cardinal clinical features are progressive, fairly symmetric muscle weakness accompanied by absent or depressed deep tendon reflexes. Patients usually present a few days to a week after onset of symptoms. The weakness can vary from mild difficulty in walking to nearly complete paralysis of all extremities, facial, respiratory, and bulbar muscles.

Studies from the United States and Europe, reflecting primarily patients with AIDP, showed that GBS is associated with the following clinical features [4, 5]:

- The weakness usually starts in the legs, but it begins in the arms or facial muscles in about 10 percent of patients.
- Severe respiratory muscle weakness necessitating ventilatory support develops in 10 to 30 percent [6].
- Facial nerve palsies occur in more than 50 percent, and oropharyngeal weakness eventually occurs in 50 percent.
- Oculomotor weakness occurs in about 15 percent of patients.
- Decreased or absent reflexes in affected arms or legs are found in approximately 90 percent of patients at presentation and in all patients with disease progression [5].
- Paresthesias in hands and feet accompany the weakness in more than 80 percent of patients, but sensory abnormalities on examination are frequently mild.
- Pain due to nerve root inflammation, typically located in the back and extremities, can be a presenting feature and is reported during the acute phase by two-thirds of patients with all forms of GBS [7, 8].
- Dysautonomia occurs in approximately 70 percent of patients [9]. In a study of data from the US Nationwide Inpatient Sample during 2010-2011, manifestations of autonomic dysfunction that were more common in patients with GBS compared with controls included diarrhea/constipation, hyponatremia, bradycardia, urinary retention, tachycardia, reversible cardiomyopathy, and Horner's syndrome [10]. Severe autonomic dysfunction is important to recognize since this is occasionally associated with sudden death [11].
- The syndrome of inappropriate antidiuretic hormone secretion (SIADH), which may be due to autonomic involvement, is another complication of GBS [10, 12, 13].

Unusual features of GBS include papilledema, facial myokymia, hearing loss, meningeal signs, vocal cord paralysis, and mental status changes [14]. In addition, posterior reversible encephalopathy syndrome has been associated with GBS in adults and children, likely related to acute hypertension from dysautonomia [15,16,17].

GBS usually progresses over a period of about two weeks. By four weeks after the initial symptoms, more than 90 percent of GBS patients have reached the nadir of the disease [5]. Disease progression for more than eight weeks is consistent with the diagnosis of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).

In patients with GBS, lumbar puncture often reveals an elevated CSF protein with a normal CSF white blood cell count. This finding, known as albumin cytologic dissociation, is present in 50 to 66 percent of patients with GBS in the first week after the onset of symptoms and ≥75 percent of patients in the third week [1, 14, 18].

Nerve conduction studies (NCS) and needle electromyography (EMG) is valuable for confirming the diagnosis of GBS and for providing information about prognosis. These are also useful in classifying the variants of GBS as demyelinating or axonal [19].

- Demyelinating forms of GBS are supported by features of demyelination, including decreased motor nerve conduction velocity, prolonged distal motor latency, increased F wave latency, conduction blocks, and temporal dispersion [20].
- Axonal forms of GBS are supported by decreased distal motor and/or sensory amplitudes [20]. Transient motor nerve conduction block (i.e. reversible conduction failure) can be present [21].

Supportive care is extremely important in Guillain Barre Syndrome (GBS) since up to 30 percent of patients develop neuromuscular respiratory failure requiring mechanical ventilation [22]. In addition, autonomic dysfunction may be severe enough to require intensive care unit (ICU) monitoring [11]. Prophylaxis for deep vein thrombosis, bladder and bowel care, physical and occupational therapy, and psychological support are essential. Low molecular weight heparin and support

stockings are recommended until patients can walk independently [22]. Bedbound patients should have frequent repositioning to avoid pressure ulcers. Adequate pain control is necessary.

Vigilance is essential when caring for a patient with GBS, since deterioration due to progression of muscle weakness can occur rapidly.

Respiratory failure in GBS is common and 15 to 30 percent of patients need ventilatory support. Thus, close respiratory monitoring with frequent measurement (e.g. every four hours) of vital capacity and negative inspiratory force (NIF) should be instituted initially in all patients [22]. Bulbar dysfunction with swallowing problems and inability to clear secretions may add to the need for ventilatory support.

The following parameters warn of impending respiratory arrest and are an indication for intubation [23]:

- Forced vital capacity <20 mL/kg
- Maximum inspiratory pressure <30 cmH_2O
- Maximum expiratory pressure <40 cmH_2O

Specific clinical findings and measurements on admission predict a high risk for respiratory failure. In a French prospective study of 722 patients with GBS not ventilated at admission, mechanical ventilation was needed in 313 (43 percent) [24]. The following factors were identified as predictors of respiratory failure:

- Time of onset to admission less than seven days
- Inability to cough
- Inability to stand
- Inability to lift the elbows
- Inability to lift the head
- Liver enzyme increases

In patients having at least four of these six predictors, mechanical ventilation was required in >85 percent.

In the subgroup of 196 patients whose vital capacity was measured on admission, three factors predicted mechanical ventilation [24]:

- Time from onset to admission less than seven days
- Inability to lift the head
- Vital capacity <60 percent of normal predicted mechanical ventilation

In patients with all three of these predictors, mechanical ventilation was required in 85 percent.

Another report of 397 patients with GBS found that independent predictors of the need for mechanical ventilation on admission were as follows [25]:

- Fewer days between onset of weakness and admission
- Presence of facial or bulbar weakness
- Severe muscle weakness

Severe autonomic disturbances occur in about 20 percent of patients, mostly (but not always) in patients who develop severe weakness and respiratory failure.

Consequently, close monitoring of blood pressure, fluid status, and cardiac rhythm is essential for the management of patients with GBS.

The main modalities of therapy for Guillain Barre Syndrome (GBS) are plasma exchange (also called plasmapheresis) and administration of intravenous immune globulin (IVIG). These treatments hasten recovery from GBS, as shown in randomized controlled trials. Patients recover sooner when treated early. The beneficial effects of plasma exchange and IVIG are believed to be equivalent, while combining the two treatments is not beneficial [26].

The choice between plasma exchange and IVIG is dependent on local availability and on patient preference, risk factors and contraindications. Plasmapheresis is thought to remove circulating antibodies, complement and soluble biological response modifiers. The precise mechanism of action for IVIG in GBS is unknown but may include providing anti-idiotypic antibodies, modulating expression and function of Fc receptors, interfering with activation of complement and

production of cytokines, and interfering with activation and effector functions of T and B cells [27, 28, 29].

Apart from plasma exchange and IVIG, no other pharmacologic agents have been found to be effective for GBS [30]. In particular, glucocorticoids are not beneficial [31, 32]. In a systematic review and meta-analysis of six trials with 587 participants, glucocorticoid-treated patients with GBS showed no significant difference in disability grade compared with patients who were not treated with glucocorticoids [33]. Furthermore, in four small trials, patients treated with oral glucocorticoids had significantly less improvement than patients who were not treated with oral glucocorticoids.

Intravenous immunoglobulin is given for five days at 0.4 gram/kg per day. Side effects include aseptic meningitis, rash, acute renal failure (mostly related to sucrose containing products), and (rarely) hyperviscosity leading to stroke. IgA deficiency can lead to anaphylaxis [29].

Factors associated with a poor prognosis for recovery from GBS include [34-38]:

- Older age
- Rapid onset (less than seven days) prior to presentation
- Severe muscle weakness on admission
- Need for ventilatory support
- An average distal motor response amplitude reduction to <20 percent of normal
- Preceding diarrheal illness

Electrodiagnostic studies can have prognostic value, especially when repeated over the first five weeks. Axonal degeneration and poor prognosis (i.e. slower recovery and/or severe residual disability) are suggested by markedly reduced distal motor response amplitude (<20 percent of normal) and profuse fibrillation potentials on needle examination, starting at two to four weeks after disease onset [39,40]. In contrast, demyelination and a good prognosis are associated with a

pattern characterized by preservation of the distal motor response amplitude above 20 percent of normal.

The proportion of patients with GBS who walk independently at six months and 12 months after diagnosis is approximately 80 and 84 percent, respectively [37]. At one year, full recovery of motor strength occurs in about 60 percent of patients, while severe motor problems persist in about 14 percent. Approximately 5 to 10 percent of patients with GBS have a prolonged course with several months of ventilator dependency and very delayed and incomplete recovery [41].

Within one year of diagnosis, approximately 3 to 7 percent of patients with GBS die despite intensive care [37, 38, 42]. Of patients who become ventilator dependent, about 20 percent will die. Causes of death include acute respiratory distress syndrome, sepsis, pulmonary emboli, and unexplained cardiac arrest [43]. Mortality appears highest in the recovery phase after neurologic improvement [38].

So, early prediction of mechanical ventilation in GBS patients is of great importance to enable physicians individualize supportive treatment. As respiratory failure contributes to significant morbidity and mortality in patients with GBS, monitoring of lung functions, anticipation of mechanical ventilation and pulmonary rehabilitation are important for optimal treatment and recovery of these patients.

AIMS AND OBJECTIVES

1. To assess involvement of Phrenic nerve using non- invasive Phrenic nerve conduction studies in LGBS patients.
2. To study various types of conduction abnormalities in Phrenic Nerve electrophysiological studies in LGBS patients.
3. To see if Phrenic Nerve conduction abnormalities have a role in predicting onset of impending respiratory failure in LGBS patients.

REVIEW OF LITERATURE

Guillain Barre Syndrome is the most common and most severe acute paralytic neuropathy. It is a clinically suspected diagnosis but nerve conduction studies can help support diagnosis to discriminate between axonal and demyelinating subtypes and could relate to prognosis.

Mortality in GBS due to ventilatory failure is around 20%. Early recognition and timely institution of appropriate measures result in considerable reduction of mortality. Clinical assessment and vital capacity measurement though useful are not sensitive enough to detect ventilatory failure in early stages. Certain studies also showed that phrenic nerve conduction studies provided the earliest indication of involvement of respiratory muscles and in most instances heralded ventilatory insufficiency.

GBS causes progressive respiratory muscle weakness involving both the inspiratory and the expiratory muscles. The weakness of the diaphragm is thought to be caused by phrenic nerve demyelination. The respiratory pattern is restrictive, as expected in a neuromuscular disease: vital capacity (VC) and total lung capacity (TLC) are diminished, the residual volume (RV) usually is normal or increased, and the RV/TLC ratio is often high in the absence of airway obstruction [44].

In a study by Barun Kumar Sen and Alak Pandit [45], abnormal phrenic nerve conduction studies were seen in 65.62 % cases (42 out of 64 patients) and among these cases, 45.24 % (19 out of 42 patients) developed respiratory failure. 7 out of 19 patients on admission had respiratory failure and 12 out of 19 developed respiratory failure during hospital stay. They also found that Single Breath Count was significantly low in those patients who developed respiratory failure. With normal phrenic nerve study, no one developed respiratory failure (NPV- 100%) and with abnormal phrenic nerve conduction study, almost half patients developed respiratory failure and all those who developed respiratory failure had abnormal phrenic nerve study (sensitivity- 100%). Thus patients with GBS with normal phrenic nerve

conduction study are unlikely to develop significant respiratory failure. Hence phrenic nerve conduction study may be considered independently in predicting respiratory failure. In phrenic nerve conduction studies they showed that its sum latency, amplitude, duration and area were abnormal in those who developed respiratory failure. It was found that age, gender, preceding infection, autonomic involvement and types of GBS did not influence development of respiratory failure. Rapid disease progression to peak disability, more severe disease, lesser single breath counts and cranial nerve involvement were seen more in patients with respiratory involvement.

In GBS, 14 – 44% of patients require respiratory support during the course of their illness [46, 47], with a reported mortality of 5 –10% and mostly due to autonomic failure and respiratory failure [48]. As compared to the West, reports from India show a higher mortality and a more fulminant form of the disease [49, 50].

Respiratory failure in GBS is mostly due to diaphragmatic weakness; however, it may be due to the involvement of accessory muscles of respiration, intercostal and abdominal muscle weakness, retained airway secretions, atelectasis, and supine posture [51].

Early recognition of respiratory failure in GBS patients is very important as they may benefit from intensive monitoring, early and optimal treatment of neuromuscular respiratory failure. Arterial blood gas analysis, spirometry, particularly forced vital capacity (FVC), and clinical assessments are relatively insensitive methods of detecting respiratory failure, particularly in early progressive stages of GBS.

Only a few studies have evaluated the role of electrophysiology of the phrenic nerve in early stage of GBS patients to detect respiratory failure [52, 53].

Clinical assessment and vital capacity measurements, though useful, are not sensitive enough to detect ventilation failure in the early stages as found in Ito *et al*.'s study [53] where clinical assessment was not correlated with phrenic nerve conduction or subsequent respiratory failure, but phrenic nerve conduction studies were proportional to measurements of respiratory functions such as vital capacity and

provided the earliest indicator of involvement of respiratory muscles [54,55].

Multiple clinical parameters have been found to serve as predictors of mechanical ventilation from different studies, including time from onset to admission of <7 days, inability to cough and stand, inability to lift the elbows or head from the bed, cranial nerve deficits, increased liver enzyme levels, anti- GQ1b antibodies, low vital capacity, phrenic nerve compound muscle amplitude potential (CMAP) latency, and the proximal/distal compound muscle amplitude potential (p/d CMAP) ratio of the peroneal nerve [24, 56-61].

Davis [62] described a simple surface recording method of phrenic nerve conduction study in humans and confirmed that it represented the true diaphragmatic muscle action potential. According to their study, clinical assessment and vital capacity measurement though useful, are not sensitive enough to detect ventilatory failure in the early stages. Theoretically, phrenic nerve conduction studies could be used as a marker of respiratory muscle weakness.

In a study by Gourie-Devi and Ganapathy [63] abnormalities in phrenic nerve conduction time were observed in 18 out of 28 patients (64.3%) at admission or on subsequent examination and 83.3% of patients with prolonged phrenic nerve conduction time developed ventilatory failure during the course of the illness. Conduction time was found to be prolonged in 18 (64.3%) patients and serial studies showed progressive improvement with restoration of normal values in majority by 12 weeks. These conduction abnormalities had a positive correlation with the extent of the disease, morbidity and mortality. Phrenic nerve conduction time was found to be more sensitive parameter than vital capacity or median nerve motor conduction velocity in assessing the severity of the disease and predicting impending ventilatory failure.

Gourie-Devi and Ganapathy [63] also reported that the phrenic nerve latency is useful in predicting disease severity, morbidity, and mortality in patients with Guillain-Barre syndrome [63] but other electrophysiological features of the respiratory system have not been

studied. We have further developed the technique of phrenic nerve conduction study and needle EMG of the diaphragm, and found them valuable in the investigation of neuromuscular respiratory failure [64]. Hence, the present work aims to describe the range of electrophysiological abnormalities of the respiratory system in patients with Guillain Barre Syndrome, and to determine the predictive value of these relevant electrophysiological studies.

Clinical assessment and vital capacity measurement though useful, are not sensitive enough to detect ventilatory failure in the early stages [62]. From the report of Newsom Davies [62] it is seen that phrenic nerve conduction studies provided the earliest indication of involvement of respiratory muscles and in most instances heralded ventilatory insufficiency. In clinical practice this warning was used to advantage in selection of patients who might require respiratory assistance in future. In their series 83.3% of patients with prolonged phrenic nerve conduction time developed ventilatory failure during the course of the illness. Phrenic nerve conduction was found to be abnormal in 64.3% (18 of 28 patients) while decrease in median motor conduction velocity was seen only in 46.4% (13 of 28 cases). In no single case was abnormal median nerve conduction velocity with normal phrenic nerve conduction noted, indicating the sensitivity and the diagnostic importance of phrenic nerve conduction.

Phrenic nerve conduction abnormality was found to be of value in predicting the severity of the disease and prognostication in terms of morbidity and mortality. The disease process was more severe and final outcome poorer in the patients with delayed conduction compared to those with normal conduction.

It is of interest that the conduction abnormality persisted up to a maximum of five months after the onset of illness and full clinical recovery had preceded the electrophysiological recovery. These findings are in agreement with the observations of other workers who noted persistence of decrease in conduction velocity of peripheral limb nerves in Guillain Barre Syndrome even after full clinical recovery [65].

Another study by Basiri et al [66] showed that compound muscle action potential (CMAP) latencies, right diaphragmatic CMAP amplitude and diaphragmatic CMAP duration were significantly different between GBS patients with respiratory failure and without respiratory failure concluding that not only phrenic nerve CMAP latency and amplitude but also diaphragmatic CMAP duration could have predictive value for respiratory failure in GBS patients.

Early prediction of mechanical ventilation is important in that 60 % of patients with mechanical ventilation developed major complications including pneumonia, sepsis, gastrointestinal bleeding. So early identification and intervention may reduce the risk of complications and improve the outcome of patients. A study by Xiujuan Wu et al [67] revealed that shorter interval from onset to admission, facial nerve palsy, glossopharyngeal and vagus nerve deficits and lower MRC sum score were risk factors for mechanical ventilation.

According to a study by Hugh J Willison et al [19], the importance of NCV is:

• Can be helpful in clinical practice, but are generally not required to diagnose Guillain Barre Syndrome.

• Needed to meet all Brighton criteria for Guillain Barre Syndrome [5].

• Essential for classification of Guillain Barre Syndrome in acute inflammatory demyelinating polyneuropathy or acute motor axonal neuropathy.

• Acute inflammatory demyelinating polyneuropathy: features of demyelination (decreased motor nerve conduction velocity, prolonged distal motor latency, increased F-wave latency, conduction blocks, and temporal dispersion) [20].

• Acute motor axonal neuropathy: no features of demyelination (one demyelinating feature in one nerve, if distal CMAP amplitude is less than 10% LLN, can be found; distal CMAP amplitude less than 80% LLN in at least two nerves [20]. Transient motor nerve conduction block might be present [21].

Durand and colleagues [59] found a significant association between respiratory failure and neurological impairment of inferior limbs. The

authors showed that peripheral neurophysiological testing was helpful for assessing the risk of respiratory failure.

According to study published in The Lancet, the risk of respiratory failure is associated with the rate of disease progression, severity of limb weakness, peroneal nerve conduction block and low vital capacity [19].

Patients with primary demyelinating neuropathy were likely to fare better in terms of outcome. In GBS, inexcitability of nerves could be due to severe conduction blocks or axonal pathology. This is the basis for analyzing the data using compound muscle action potential amplitudes [68].

Patients with inexcitable nerves had a poor outcome at discharge. Axonal degeneration is independently associated with more severe disease and less chance of recovery [69].

In our study, we are going to assess the importance of phrenic nerve conduction studies in predicting respiratory abnormalities and the various abnormalities observed in phrenic nerve conduction studies. Few bedside parameters like single breath count and power in all limbs were also compared for any advantage in predicting respiratory involvement in GBS patients. Additionally, patient factors were also compared. Peripheral motor nerve conduction abnormalities were also observed and compared if they could predict the progression of disease as respiratory involvement.

MATERIALS AND METHODS

All patients admitted to DMCH Ludhiana under the Department of Medicine and the Department of Neurology from 1st November 2018 to 30th April 2020 fulfilling the inclusion criteria and not falling in the exclusion criteria, and giving consent for being enrolled in our study were included.

All patients admitted to DMCH Ludhiana with suspicion of having LGBS with complaints of:

- leg and arm weakness (32%) or
- selective proximal and distal leg weakness (56%) often spreading to the arm or
- weakness in arm or
- symmetric ascending paralysis or
- paraparesis or
- quadriparesis or
- difficulty bearing weight on both legs or
- Acroparesthesia

who were suspected to have GBS were taken in our study after written consent and NCV studies including Phrenic Nerve studies were performed on them.

Definition of Single Breath Count (SBC): Measurement of how far an individual can count in a normal speaking voice after a maximal effort inhalation.

<u>DETAILS OF NCV STUDIES TO BE PERFORMED:</u>

Active electrodes are placed on the center of muscle belly with reference electrodes being placed over muscle tendon distally. Ground electrodes are also placed between active and reference electrodes. Stimulator for stimulating the nerve under study is placed over the nerve that supplies the muscle. It has a cathode (negative pole) that

depolarizes the underlying nerve segment and an anode (positive pole) that hyperpolarizes underlying nerve segment.

As the nerve is stimulated, a compound muscle action potential (CMAP) is generated which represents the summation of all underlying individual muscle fibre action potentials. The following parameters of this muscle action were studied:

- Latency: time interval between onset of stimulus and onset of response
- Amplitude: maximum height of action potential
- Conduction Velocity: It is the speed of propagation of an electrical impulse through the nerve calculated as distance travelled by the impulse per unit time.
- F-wave latency: It is the shortest latency to onset of the initial deflection (either negative or positive) of all recorded F waves which are compound action potentials evoked by supra-maximal antidromic stimulation of a motor nerve.
- F-wave persistence: The number of occurrences of response divided by number of stimuli which is a measure of antidromic excitability of a particular motor neuron pool.

Table 1- Electrodes placement					
			Recording electrodes		Stimulation Sites
			Active Electrode (G1)	Reference Electrode (G2)	
Upper Limb	Median Nerve	Motor	Abductor pollicis brevis muscle belly	First metacarpo-phalangeal joint	1. Middle of wrist between tendons of FCR and Palmaris longus 2. Antecubital fossa over brachial artery pulse
		Sensory	Index/ middle finger Metacarpo-phalangeal joint	3-4 cm distally over DIP joint	Middle of wrist between tendons of FCR and Palmaris longus
	Ulnar Nerve	Motor	Abductor digiti minimi muscle belly	Fifth metacarpo-phalangeal joint	1. Medial wrist adjacent to FCU tendon 2. Elbow 3cm distal to medial epicondyle
		Sensory	Little finger metacarpo-phalangeal joint	3-4 cm distally over DIP joint	Medial wrist adjacent to FCU tendon
Lower Limb	Peroneal Nerve	Motor	Dorsal lateral foot over Extensor digitorum brevis muscle belly	Metatarso-phalangeal joint of little toe	1.Ankle anteriorly slightly lateral to tibialis anterior tendon 2. Head of fibula
	Tibial Nerve	Motor	1 cm proximal and 1 cm inferior to navicular prominence over Abductor hallucis brevis muscle belly	Metatarso-phalangeal joint of great toe	1.Medial ankle- slightly proximal and posterior to medial malleolus 2. Popliteal fossa- Mid-posterior knee over popliteal pulse
	Sural Nerve	Sensory	Posterior ankle posterior to lateral malleolus	3-4 cm distally at posterior ankle	Posterior lateral calf
Phrenic Nerve		Motor	Two fingerbreadths (5cm) above xiphoid process over diaphragm muscle	Anterior costal margin 16 cm from G1	1.Lateral neck posterior to sternocleidomastoid muscle, approximately 3 cm above the clavicle 2. Anterior lateral neck between sterna and clavicular heads of SCM, just above the clavicle

Table 2- Normal values						
	Nerve	Record	Amplitude	Conduction Velocity	Onset (for Motor) / Peak (for Sensory) Latency	Minimal F-Wave Latency
Upper Limb	Median nerve	Abductor policis brevis	>5.0 mV	>45 m/s	<4.3 ms	<31.2 ms
		Sensory	>11 microV	≥50 m/s	<3.6 ms	
	Ulnar nerve	Abductor digiti minimi	>5.5 mV	>43 m/s	<3.5 ms	<32.1 ms
		Sensory	>11 microV	≥50 m/s	<3.0 ms	
Lower Limb	Peroneal nerve	Extensor digitorum brevis	>2.0 mV	≥37 m/s	<5.4 ms	<59.3 ms
	Tibial nerve	Abductor hallucis brevis	>3.5 mV	>35 m/s	<6.1 ms	<54 ms
	Sural nerve	Sensory	>9.0 microV	≥40 m/s	<3.1 ms	
Phrenic nerve		Diaphragm	597 ± 139 (>320) microV		6.3 ± 0.8 (<8.0) ms	

F-WAVE PERSISTENCE-

MEDIAN NERVE- >50 %

ULNAR NERVE- >50 %

PERONEAL NERVE- >30 %

TIBIAL NERVE- >50 %

Any deviation from these normal values was studied and compiled and results were charted. These patients were followed up till their stay in hospital for development of any type of respiratory involvement and then correlation was made for any abnormalities in Phrenic Nerve conduction studies done during admission and results were then charted.

SOURCE OF DATA

Patients from DMCH Ludhiana admitted in Department of Medicine and Department of Neurology of DMCH Ludhiana with clinical suspicion of LGBS were included in this study from 1st November 2018 to 30th April 2020 and NCV studies including Phrenic Nerve were performed on them and patients were followed up till their stay in hospital.

METHOD OF COLLECTION OF DATA

All patients were admitted to DMCH Ludhiana with suspicion of having LGBS. Then NCV studies including Phrenic Nerve were performed on such patients to make the diagnosis of LGBS. Patients were followed up till their hospital stay for any respiratory abnormalities and then these were correlated with any Phrenic Nerve electrophysiological changes observed during NCV done during admission.

Phrenic nerve conduction studies were done with patient being supine with head slightly elevated and rotated to the side opposite to the nerve under stimulation. The phrenic nerve was stimulated percutaneously in the neck at the posterior border of sternocleidomastoid at the level of upper margin of thyroid cartilage. Then surface recording electrodes

were placed in 8^{th}/ 9^{th} intercostals space with anterior electrodes in anterior axillary line.

All patients fulfilling the inclusion criteria were included in the study and patients falling in exclusion criteria were excluded.

INCLUSION CRITERIA

1. Age ≥ 18 years
2. Clinically suspected cases of LGBS
3. Diagnosis made on the basis of non-invasive electrodiagnostic studies i.e. NCV studies.

EXCLUSION CRITERIA

1. Other predisposing factors of polyneuropathy:
   i) Known case of Diabetes Mellitus
   ii) Known case of Hypothyroidism

NUMBER OF PATIENTS

All patients giving consent for enrolling in our study admitted to DMCH Ludhiana in the Department of Medicine and the Department of Neurology from 1^{st} November 2018 to 30^{th} April 2020, fulfilling the inclusion criteria but not falling under exclusion criteria were considered for our study.

OUTCOME

Various abnormalities in Phrenic Nerve conduction studies were observed in LGBS diagnosed cases and their role in giving an insight into the possibility of patient developing some respiratory distress was studied.

STATISTICAL ANALYSIS

Data was described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups was done using Student t-test and Mann Whitney *U* test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi

square ($\chi 2$) test was performed and exact test was used when the expected frequency is less than 5. A probability value (*p* value) less than 0.05 was considered statistically significant. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

Landry Guillain Barre Syndrome (LGBS) is an acute immune-mediated polyneuropathy, maybe provoked by a preceding infection. Our study enrolled 47 patients who were more than or equal to 18 years of age, with clinical suspicion of LGBS who presented to us with myriad of symptomatology with either symmetrical or asymmetrical weakness of limbs. These symptoms were usually acute in onset and progressive over hours to days upto 4 weeks. These patients were admitted to DMCH Ludhiana either through emergency or OPD. On the basis of symptomatology and clinical evaluation, possibility of LGBS was kept and NCV studies were done which if showed evidence of LGBS, Phrenic nerve conduction studies were then performed on them excluding those patients who were known diabetics or had prior hypothyroidism. Various clinical and electrophysiological parameters were considered and compared among patients who had respiratory involvement during hospital stay and who had no respiratory involvement.

Amongst the total of 47 cases, 29.8% (14 patients) had respiratory involvement and 64.3% (9 out of 14) of them required mechanical ventilation.

Table 3- Respiratory involvement seen

Respiratory involvement	No. of cases	Percentage
Absent	33	70.2%
Present	14	29.8%
Total	47	100.0%

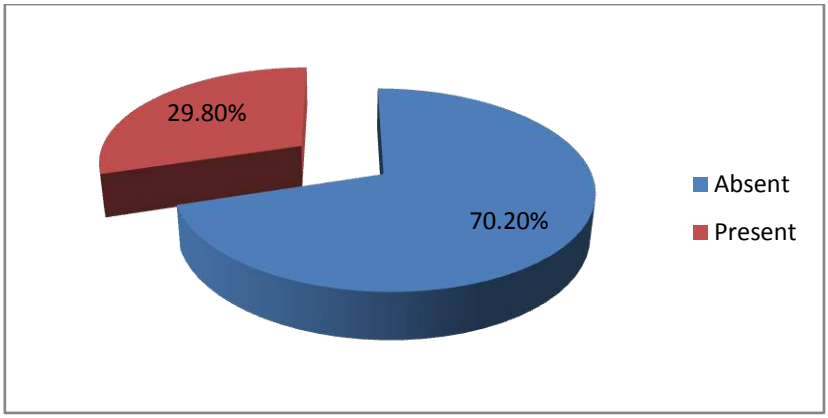

Figure 1- Respiratory involvement seen

Table 4- Patients who required mechanical ventilation		
Need for mechanical ventilation	No. of cases	Percentage
No	38	80.9%
Yes	9	19.1%
Total	47	100.0%

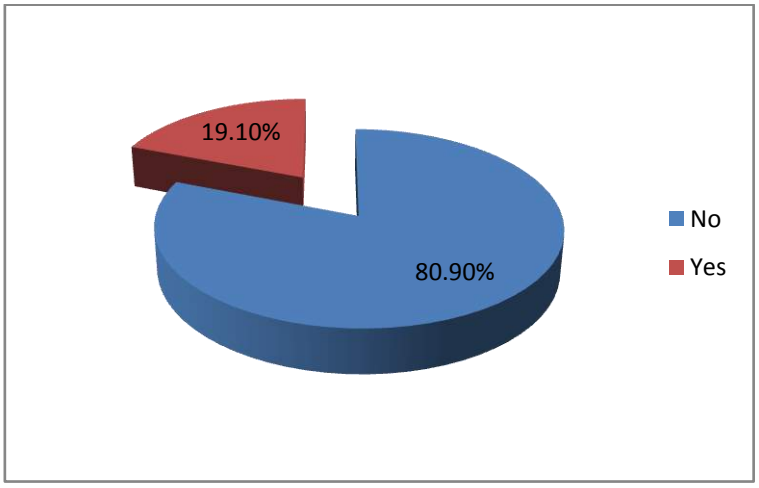

Figure 2- Patients with severe disease requiring mechanical ventilation

Table 5- Patients with respiratory involvement progressing to mechanical ventilation								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Need for mechanical ventilation	No	33	100.0%	5	35.7%	38	26.239	0.0001
	Yes	0	0.0%	9	64.3%	9		
Total		33	100.0%	14	100.0%	47		

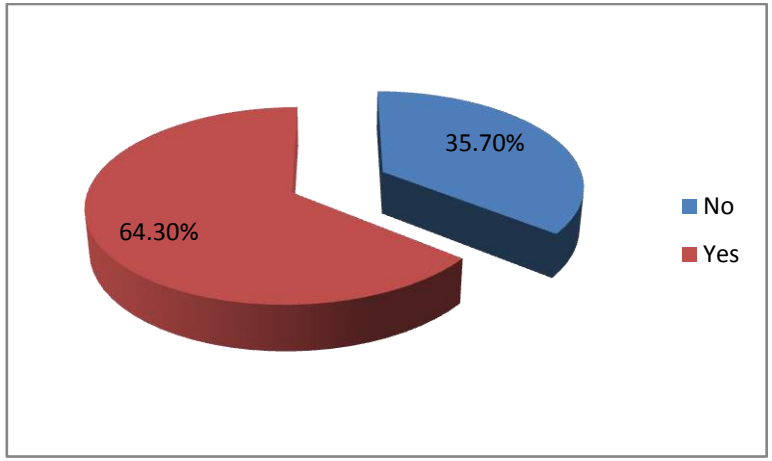

Figure 3- Patients with respiratory involvement progressing to mechanical ventilation

AGE AND GENDER DISTRIBUTION

In our study, 44.7% (21 patients) patients were 31 to 50 years of age, 25.5% (12 patients) were less than 30 years of age and 29.8% (14 patients) were more than 50 years of age. Fifty percent patients with respiratory involvement were 31 to 50 years of age. Mean age of patients who had respiratory involvement was 39.57 ± 16.20 years. No significant co-relation of respiratory involvement in LGBS patients with age was seen as p- values were >0.05.

Table 6- Age distribution

Age group	No. of cases	Percentage
< 30	12	25.5%
31-50	21	44.7%
> 50	14	29.8%
Total	47	100.0%

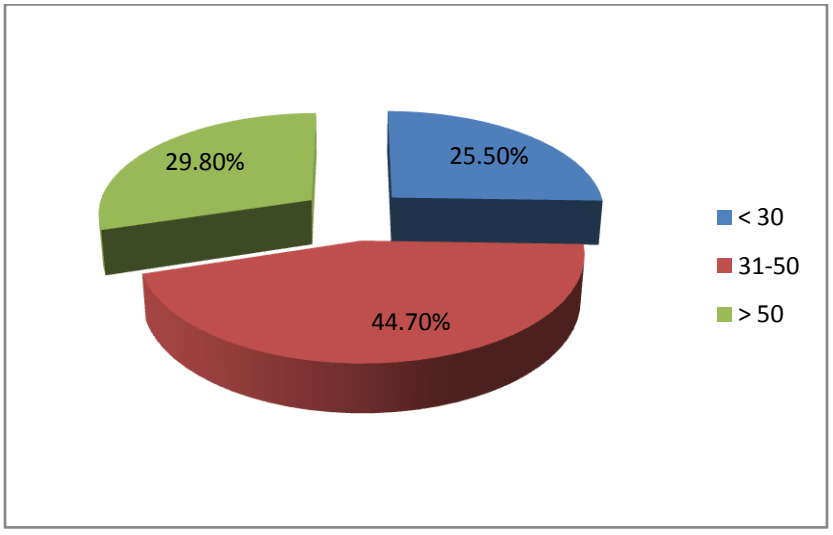

Figure 4- Age distribution

Table 7- Age differences in both groups with and without respiratory involvement						
	Respiratory involvement					
	Absent		Present		t	p-value
	Mean	SD	Mean	SD		
Age (Years)	43.85	16.99	39.57	16.20	0.800	0.428

Table 8- Influence of age on respiratory involvement in LGBS								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Age group	< 30	8	24.2%	4	28.6%	12	0.666	0.717
	31-50	14	42.4%	7	50.0%	21		
	> 50	11	33.3%	3	21.4%	14		
Total		33	100.0%	14	100.0%	47		

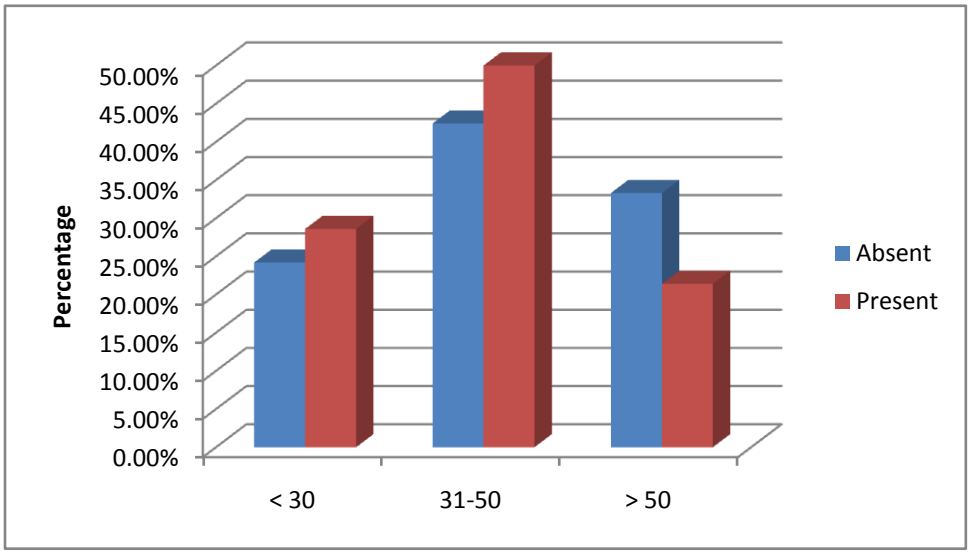

Figure 5- Respiratory involvement according to age in LGBS

Amongst 47 patients, 21 were females and 26 were males. Amongst 14 patients with respiratory involvement, 50% were males and other 50% were females showing p- value of 0.633 suggestive that gender has no co-relation with respiratory involvement in LGBS.

Table 9- Gender distribution		
Gender	No. of cases	Percentage
Female	21	44.7%
Male	26	55.3%
Total	47	100.0%

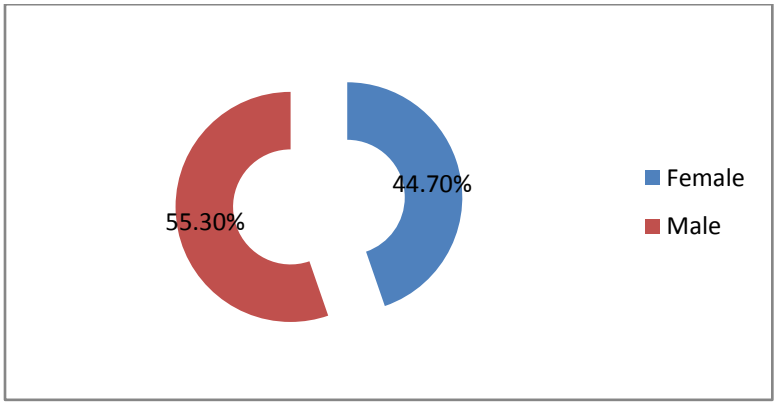

Figure 6- Gender distribution

Table 10- Gender distribution and influence on respiratory involvement in LGBS								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Gender	Female	14	42.4%	7	50.0%	21		
	Male	19	57.6%	7	50.0%	26	0.228	0.633
Total		33	100.0%	14	100.0%	47		

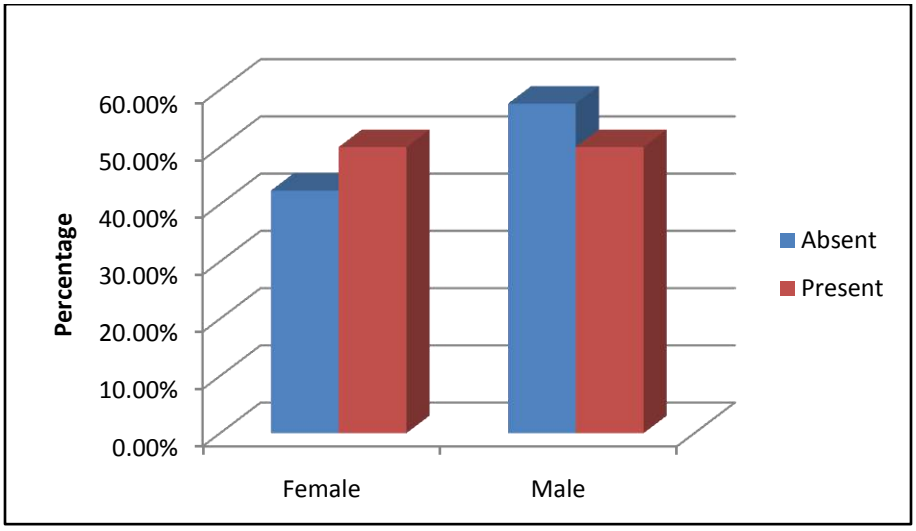

Figure 7- Influence of gender on respiratory involvement in LGBS patients

CLINICAL PARAMETERS

Patients were then clinically examined and various parameters compared. Co-relation of respiratory rate with respiratory involvement in LGBS was also studied. The co-relation was found to be significant with p-value of <0.001. The mean respiratory rate for patients without respiratory involvement was 20± 2.29 per minute whereas for patients with respiratory involvement it was 26.46± 2.18 per minute which was more in this group and it can be anticipated that more the respiratory rate on admission, more are the chances of patient progressing to respiratory distress.

Table 11- Respiratory rate on admission predicting respiratory deterioration in LGBS

	Respiratory involvement					
	Absent		Present		t	p-value
	Mean	SD	Mean	SD		
Respiratory rate (per minute) (on admission)	20.00	2.29	26.46	2.18	-8.722	< 0.001

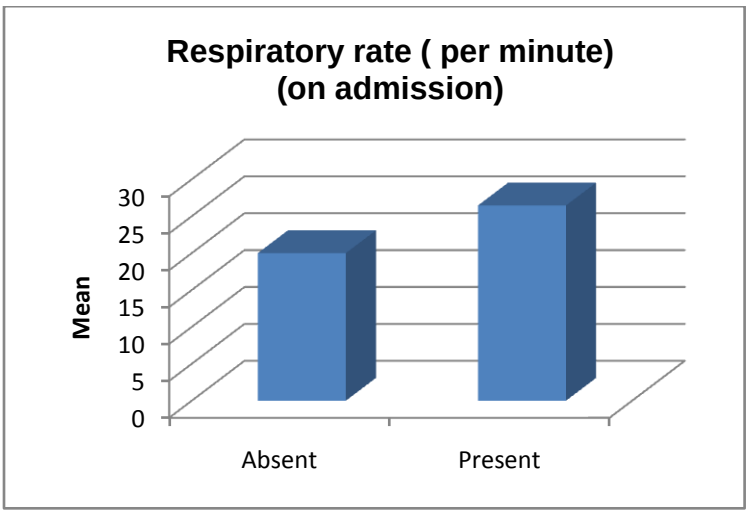

Figure 8- On admission mean respiratory rate more in those progressing to respiratory involvement

Assessment of Single Breath Count (SBC) is an important bedside clinical parameter that can be used for prediction of respiratory involvement in LGBS patients. In our study also, SBC was assessed in 46 patients as 1 patient was already on tracheostomy and so SBC could not be assessed in that patient. Patients having respiratory distress had a mean SBC of 16.38 ± 7.35 and those without respiratory involvement had a mean of 25.91 ± 11.31, giving a p- value of 0.008 suggestive of significant co-relation with SBC and respiratory involvement prediction in LGBS patients.

Table 12- Mean SBC in LGBS

	Respiratory involvement					
	Absent		Present		t	p-value
	Mean	SD	Mean	SD		
Single breath count	25.91	11.31	16.38	7.35	2.802	0.008

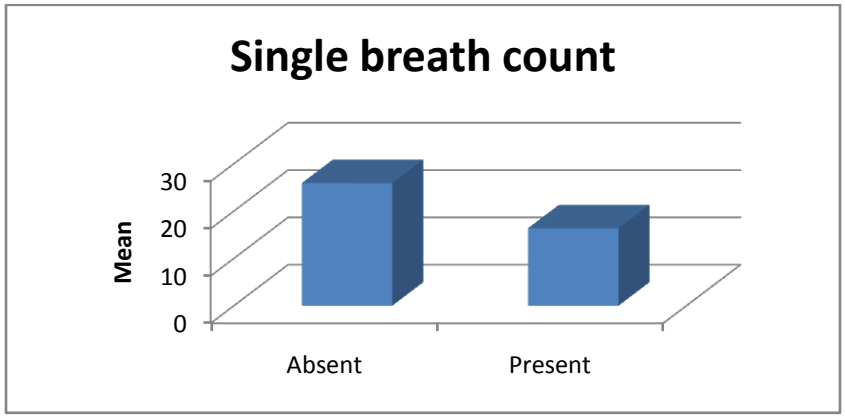

Figure- 9 Mean SBC lesser in patients with respiratory involvement

Power assessment in various muscle groups was done in all 4 limbs and power grading was divided in patients with power ≤3 and power >3, power of left shoulder and elbow and left ankle gave significant p- values (<0.05). Thus, power grade ≤3 might be suggestive of severe disease predicting respiratory involvement in LGBS patients as 78.6 % (11 out of 14) patients with respiratory involvement had power ≤ 3 in left shoulder as well as elbow as compared to 45.5 % (15 out of 33) and 39.4% (13 out of 33) patients without respiratory involvement having power grade ≤ 3 in left shoulder and elbow respectively.

Similarly, 85.7 % (12 out of 14) patients with respiratory involvement and 51.1% (17 out of 33) patients without respiratory involvement had power grade ≤ 3 in left ankle.

Unilateral significance of power might be limited in view of small sample size of our study.

Although hand grip showed insignificant p-value, it could be concluded that lesser the handgrip, more the weakness, more severe the disease which might predict respiratory progression. On both sides, 71.4% patients (10 out of 14) with respiratory involvement had hand grip <50% as compared to just 45.5% patients (15 out of 33) without respiratory involvement.

Table 13- Power grading and co-relation with respiratory involvement in LGBS									
Power (On Admission)		Respiratory Involvement				Total	Chi-square value	p-value	odd ratio (95%CI)
		Absent		Present					
Upper Limb Right_Shoulder	≤ 3	16	48.5%	10	71.4%	26	2.094	0.148	
	> 3	17	51.5%	4	28.6%	21			
Upper Limb Right_Elbow	≤ 3	14	42.4%	10	71.4%	24	3.309	0.069	
	> 3	19	57.6%	4	28.6%	23			
Upper Limb Right_Hand Grip (%)	< 50	15	45.5%	10	71.4%	25	2.664	0.103	
	> 50	18	54.5%	4	28.6%	22			
Upper Limb Left_Shoulder	≤ 3	15	45.5%	11	78.6%	26	4.362	0.037	4.40 (1.03-18.74)
	> 3	18	54.5%	3	21.4%	21			
Upper Limb Left_Elbow	≤ 3	13	39.4%	11	78.6%	24	6.038	0.024	5.64 (1.32-24.17)
	> 3	20	60.6%	3	21.4%	23			
Upper Limb Left_Hand Grip (%)	< 50	15	45.5%	10	71.4%	25	2.664	0.123	
	> 50	18	54.5%	4	28.6%	22			
Lower Limb Right_Hip	≤ 3	23	69.7%	13	92.9%	36	2.941	0.136	
	> 3	10	30.3%	1	7.1%	11			
Lower Limb Right_Knee	≤ 3	21	63.6%	12	85.7%	33	2.291	0.130	
	> 3	12	36.4%	2	14.3%	14			

Lower Limb Right_Ankle	≤ 3	17	51.5%	11	78.6%	28	2.988	0.111	
	> 3	16	48.5%	3	21.4%	19			
Lower Limb Left_Hip	≤ 3	24	72.7%	13	92.9%	37	2.378	0.123	
	> 3	9	27.3%	1	7.1%	10			
Lower Limb Left_Knee	≤ 3	21	63.6%	13	92.9%	34	4.195	0.072	
	> 3	12	36.4%	1	7.1%	13			
Lower Limb Left_Ankle	≤ 3	17	51.5%	12	85.7%	29	4.865	0.047	5.65 (1.09-29.26)
	> 3	16	48.5%	2	14.3%	18			
Total		33	100.0%	14	100.0%	47			

It can be interpreted that patients with power in left shoulder less than or equal to 3 are 4.4 times more prone to respiratory involvement as compared to patients with power >3. Similarly, patients with power in left elbow and left ankle less than or equal to 3 are 5.64 and 5.65 times more prone to have respiratory involvement in the natural course of their disease as compared to patients with power more than 3.

In all the muscle groups, percentage of patients progressing to respiratory involvement had power grade ≤3 (as shown in Figure 10 and 11) although only three muscle groups showed significant p-value.

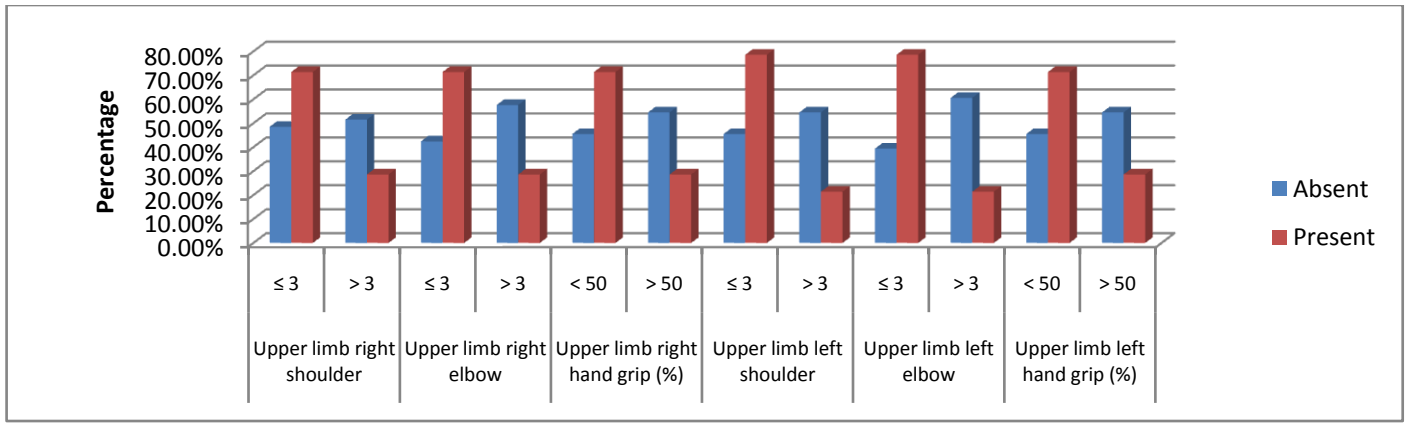

Figure 10- Power grading predicting respiratory involvement in LGBS in upper limbs

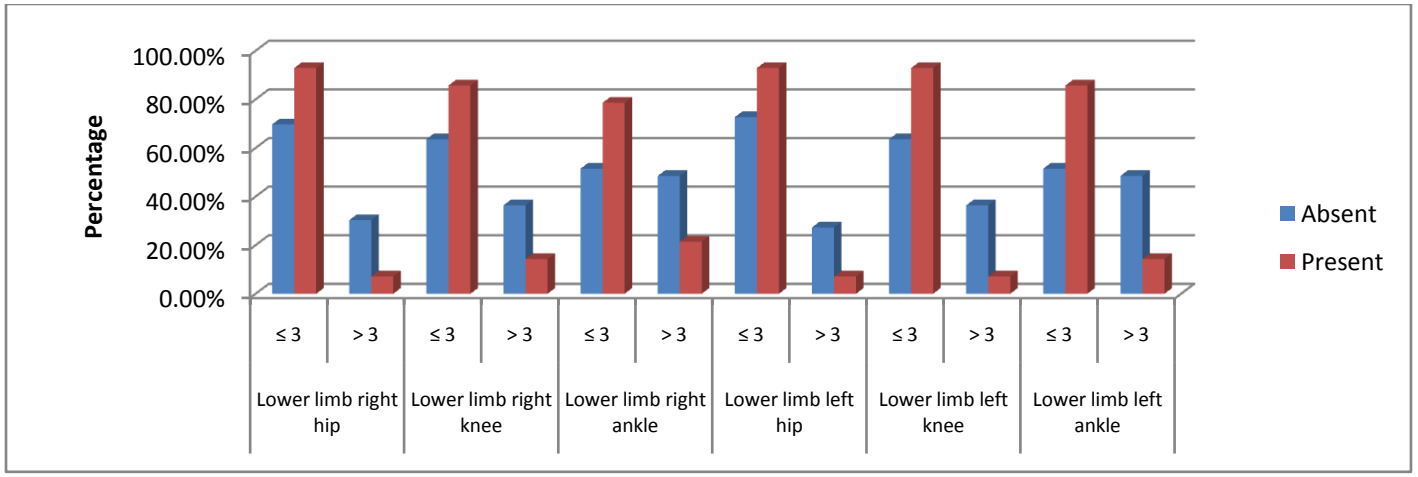

Figure 11- Power grading predicting respiratory involvement in LGBS in lower limbs

PHRENIC NERVE CONDUCTION STUDIES

Phrenic nerve conduction studies were performed in all patients in whom NCV studies were suggestive of LGBS and its amplitude and latencies were recorded.

Whenever phrenic nerve was involved in LGBS patients either with or without respiratory involvement, major phrenic nerve conduction abnormalities found were either decreased amplitude or increased latency or no response at all.

On left side, phrenic nerve amplitude was found to be decreased or showed no response in 78.7 % cases (37 patients). Among them, 29.7% (11 patients) showed respiratory involvement and p-value was not significant (p-value 0.987). Rest 70.3% (26 patients) had decreased amplitude or no response but had no respiratory involvement.

Table 14- Phrenic nerve motor amplitude on left side

Left Phrenic nerve motor amplitude	No. of cases	Percentage
Decreased	23	48.9%
No Response	14	29.8%
Normal	10	21.3%
Total	47	100.0%

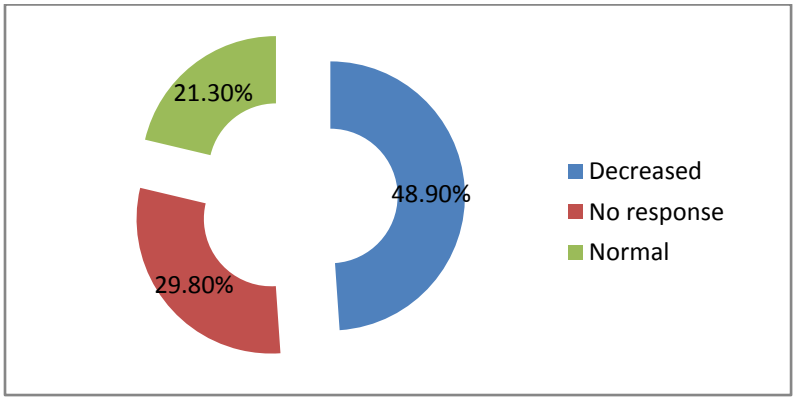

Figure 12- Left phrenic nerve motor amplitude findings

Table 15- Co-relation of left phrenic nerve motor amplitude with respiratory involvement								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Left phrenic nerve motor amplitude	Abnormal	26	70.3%	11	29.7%	37	0	0.987
	Normal	7	70.0%	3	30.0%	10		
Total		33	70.2%	14	29.8%	47		

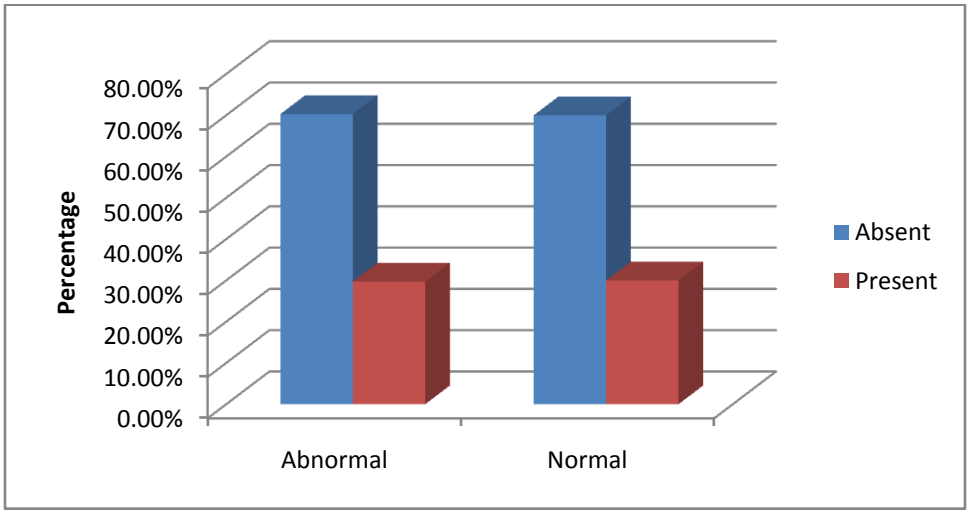

Figure 13- Left phrenic nerve motor amplitude relation with respiratory involvement in LGBS patients

On right side, phrenic nerve amplitude was found to be decreased or showed no response in 80.8 % cases (38 patients). Among them 31.6 % (12 patients) showed respiratory involvement (p-value 0.581). Rest 68.4% (26 patients) had decreased amplitude or no response but had no respiratory involvement.

Table 16- Phrenic nerve motor amplitude on right side		
Right phrenic nerve motor amplitude	No. of cases	Percentage
Decreased	29	61.7%
No Response	9	19.1%
Normal	9	19.1%
Total	47	100.0%

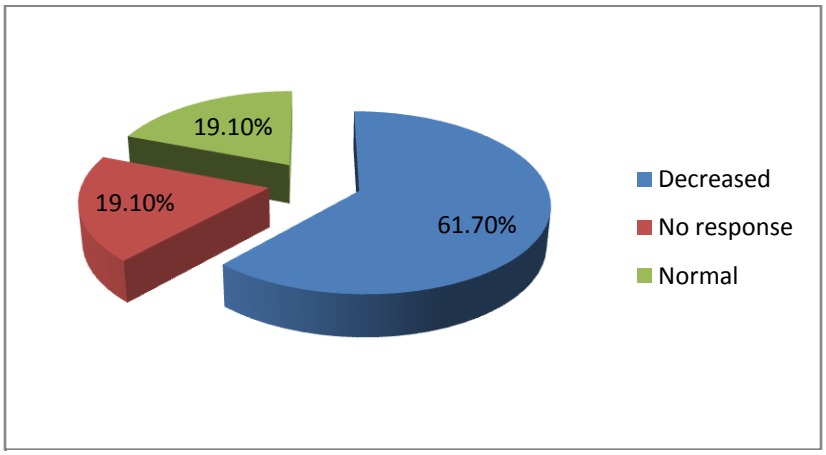

Figure 14- Right phrenic nerve motor amplitude findings

Table 17- Co-relation of right phrenic nerve motor amplitude with respiratory involvement								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Right phrenic nerve motor amplitude	Abnormal	26	68.4%	12	31.6%	38	0.305	0.581
	Normal	7	77.8%	2	22.2%	9		
Total		33	70.2%	14	29.8%	47		

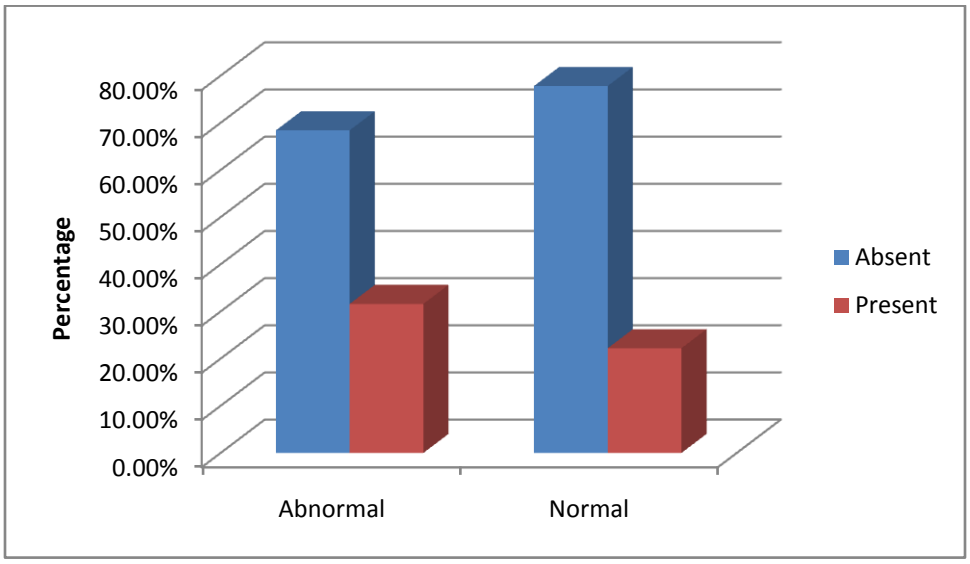

Figure 15- Right phrenic nerve motor amplitude relation with respiratory involvement in LGBS patients

No response was seen in 14 patients on left side and 9 patients on right side. Nine patients showed no response at all bilaterally and 3 of them progressed to respiratory failure.

Therefore, unilaterally, no significant co-relation was found between phrenic nerve amplitude and respiratory involvement in LGBS patients.

Bilateral involvement of phrenic nerve amplitude that is decreased amplitude or no response was seen in 78.7% cases (37 cases) with 29.7% (11 cases) having respiratory involvement. There were 3 cases amongst 14 with respiratory involvement which did not show bilateral abnormality in amplitude of phrenic nerve.

Thus, even bilateral involvement of phrenic nerve amplitude did not predict respiratory involvement in LGBS patients (p-value 0.987).

Table 18- Bilateral phrenic nerve amplitude involvement		
Amplitude bilateral involvement	No. of cases	Percentage
No	10	21.3%
Yes	37	78.7%
Total	47	100.0%

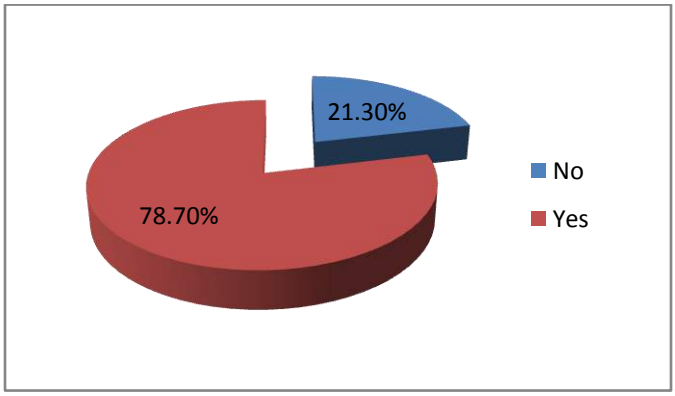

Figure 16- Bilateral phrenic nerve amplitude abnormality

Table 19- Bilateral phrenic nerve motor amplitude involvement with respiratory involvement								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Amplitude bilateral involvement	No	7	70.0%	3	30.0%	10	0.000	0.987
	Yes	26	70.3%	11	29.7%	37		
Total		33	70.2%	14	29.8%	47		

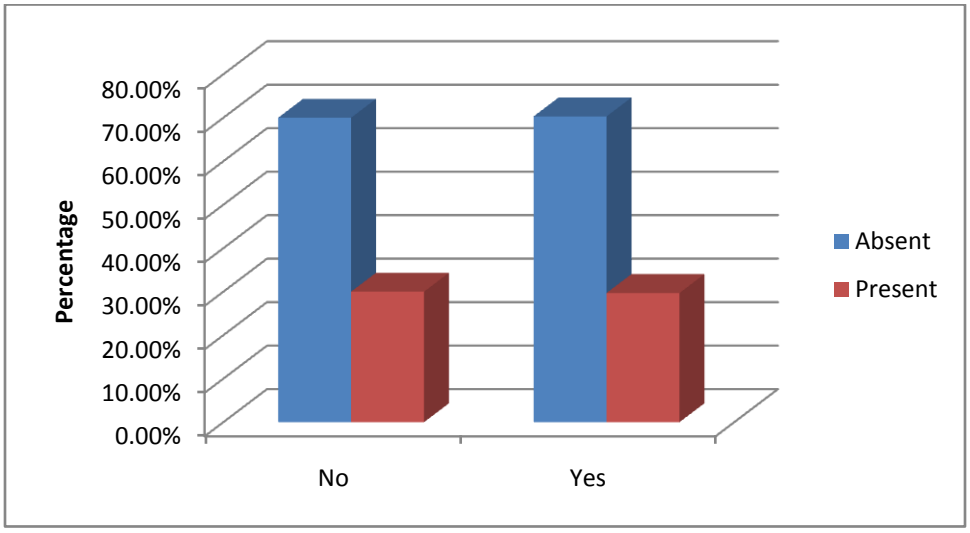

Figure 17- Bilateral phrenic nerve amplitude involvement relation with respiratory involvement in LGBS patients

During phrenic nerve conduction studies, another abnormality observed was increase in latency or no response at all.

On right side, 49 % cases (23 cases) showed increase in latency or no response at all. Among them 39.1% (9 cases) patients had respiratory involvement and 60.9% (14 cases) had no respiratory involvement. This was insignificant (p-value 0.170) for predicting respiratory progression in LGBS patients.

Table 20- Phrenic nerve motor latency on right side		
Right phrenic nerve onset latency	No. of cases	Percentage
Increased	10	21.3%
No Response	13	27.7%
Normal	24	51.1%
Total	47	100.0%

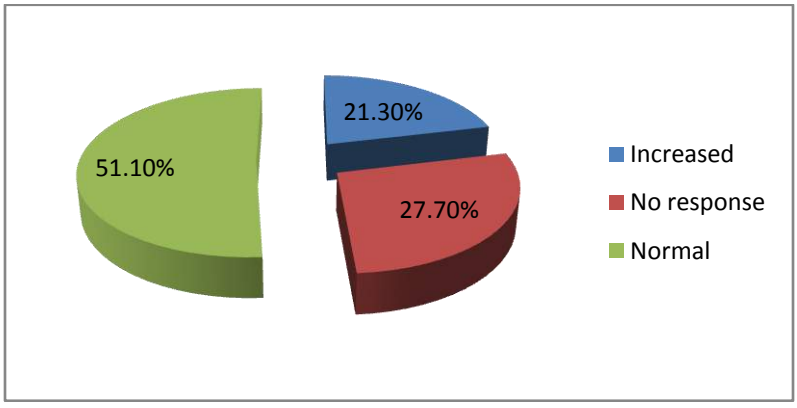

Figure 18- Right phrenic nerve latency findings

Table 21- Co-relation of right phrenic nerve motor latency with respiratory involvement								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Right phrenic nerve onset latency	Abnormal	14	60.9%	9	39.1%	23	1.88	0.170
	Normal	19	79.2%	5	20.8%	24		
Total		33	70.2%	14	29.8%	47		

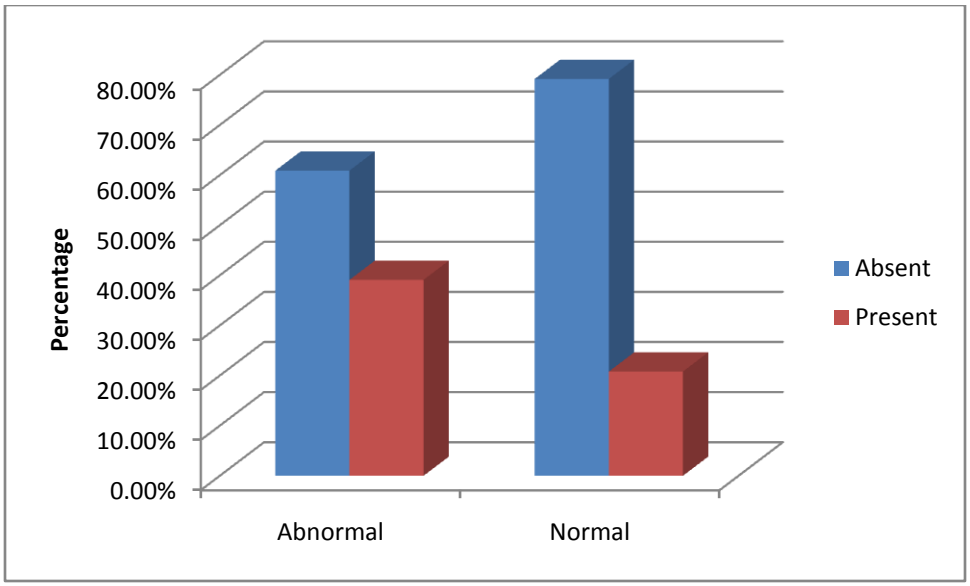

Figure 19- Right phrenic nerve motor latency relation with respiratory involvement in LGBS patients

On left side, 38.3% cases (18 cases) showed increase in latency or no response at all. Among them 38.9% (7 cases) had respiratory involvement which was again insignificant (p-value 0.282) for predicting respiratory involvement.

Table 22- Phrenic nerve motor latency on left side		
Left phrenic nerve onset latency	No. of cases	Percentage
Increased	10	21.3%
No response	8	17.0%
Normal	29	61.7%
Total	47	100.0%

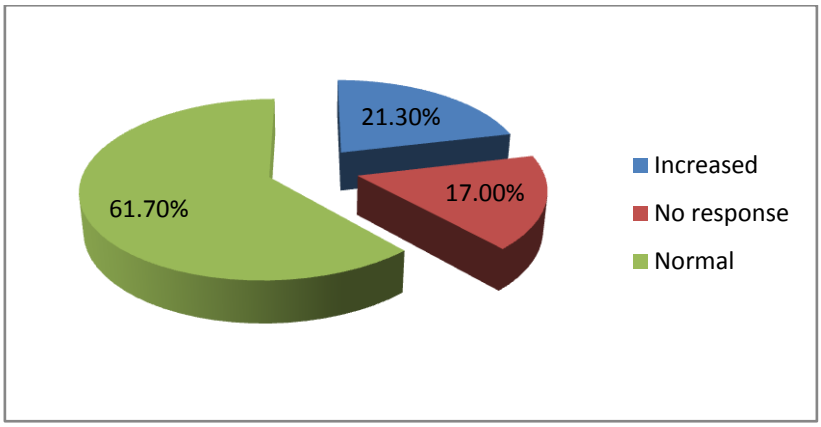

Figure 20- Left phrenic nerve motor latency findings

Table 23- Co-relation of left phrenic nerve motor latency with respiratory involvement								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Right phrenic nerve onset latency	Abnormal	11	61.1%	7	38.9%	18	1.155	0.282
	Normal	22	75.9%	7	24.1%	29		
Total		33	70.2%	14	29.8%	47		

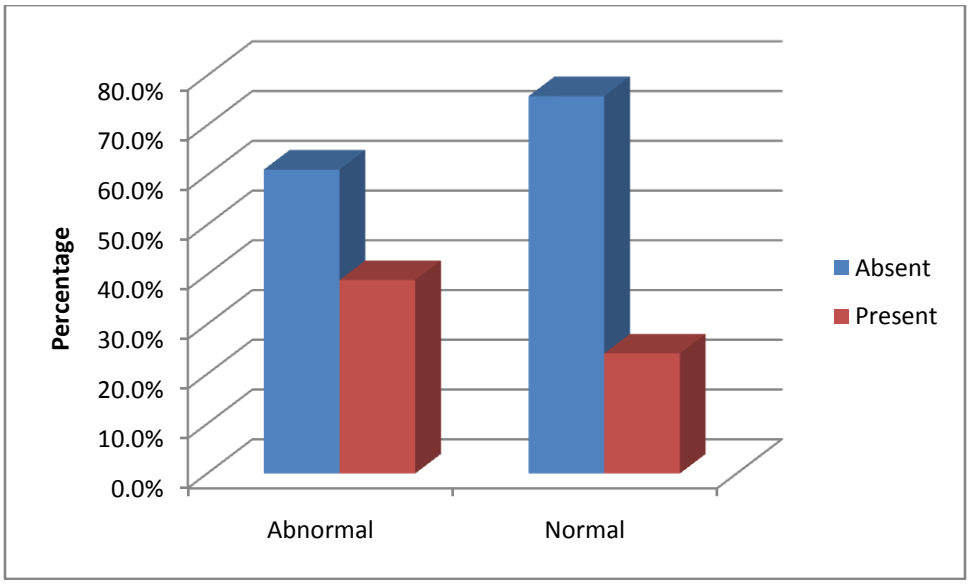

Figure 21- Left phrenic nerve motor latency relation with respiratory involvement in LGBS patients

Thirteen patients on right side and eight patients on left side showed no response. Bilateral no response was seen in 8 cases and 3 of them progressed to respiratory failure.

Therefore, unilateral involvement of phrenic nerve latency had no co-relation with respiratory involvement in LGBS patients and so bilateral latency involvement was also studied.

Bilateral involvement of phrenic nerve with increase in latency or no response at all was observed in 34% cases (16 cases). Among them 43.8% (7 cases) showed respiratory involvement with p-value not significant (p- value 0.133).

Table 24- Bilateral phrenic nerve latency involvement		
Onset latency bilateral involvement	No. of cases	Percentage
No	31	66.0%
Yes	16	34.0%
Total	47	100.0%

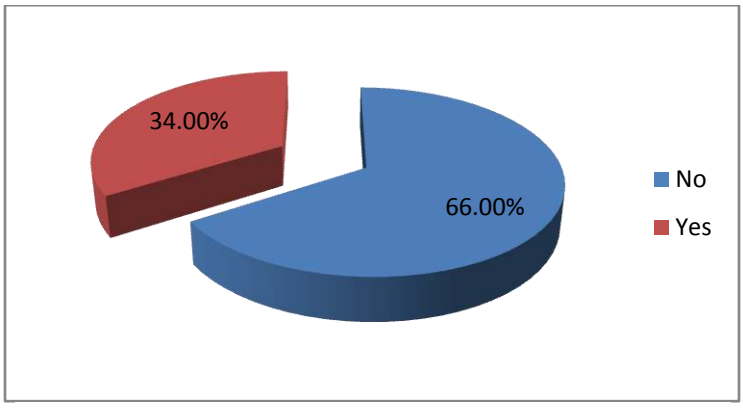

Figure 22- Bilateral phrenic nerve latency abnormality

Table 25- Bilateral phrenic nerve motor latency with respiratory involvement in LGBS								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Onset latency bilateral involvement	No	24	77.4%	7	22.6%	31	2.261	0.133
	Yes	9	56.3%	7	43.8%	16		
Total		33	70.2%	14	29.8%	47		

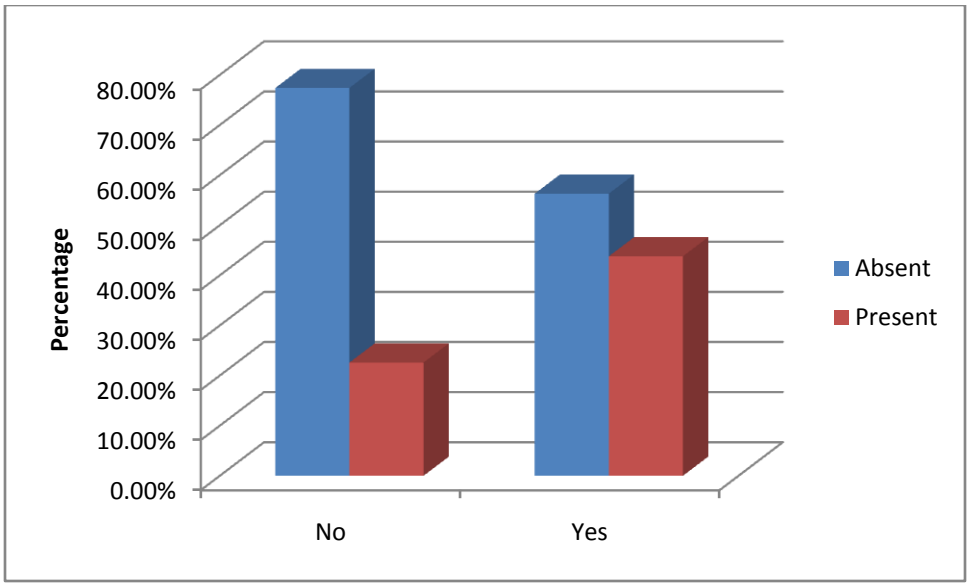

Figure 23- Bilateral phrenic nerve motor latency involvement relation with respiratory involvement in LGBS patients

Bilateral involvement of phrenic nerve amplitude and latency was seen in 27.7% (13 cases) of the total cases and among them respiratory involvement was seen in 46.2% cases (6 cases) but with an insignificant p-value (p- value 0.129).

Table 26- Bilateral phrenic nerve amplitude as well as latency involvement		
Bilateral involvement of both amplitude and latency	No. of cases	Percentage
No	34	72.3%
Yes	13	27.7%
Total	47	100.0%

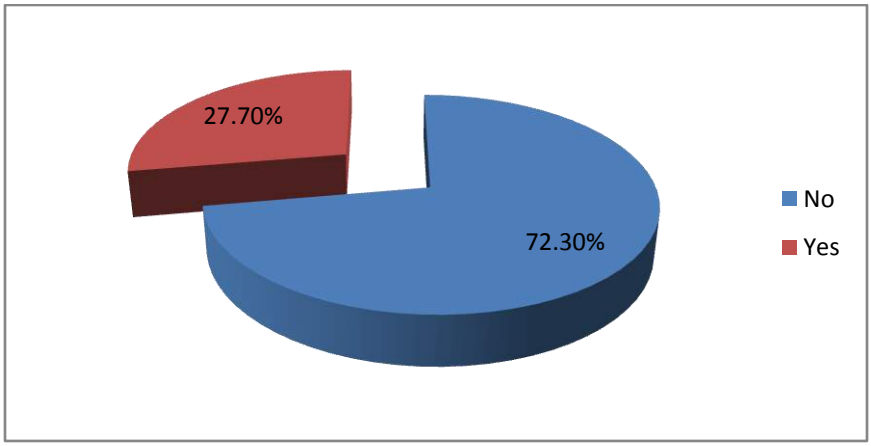

Figure 24- Bilateral phrenic nerve amplitude and latency involvement

Table 27- Bilateral phrenic nerve amplitude as well as latency involvement predicting respiratory involvement in LGBS								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Bilateral involvement of both amplitude and latency	No	26	76.5%	8	23.5%	34	2.302	0.129
	Yes	7	53.8%	6	46.2%	13		
Total		33	70.2%	14	29.8%	47		

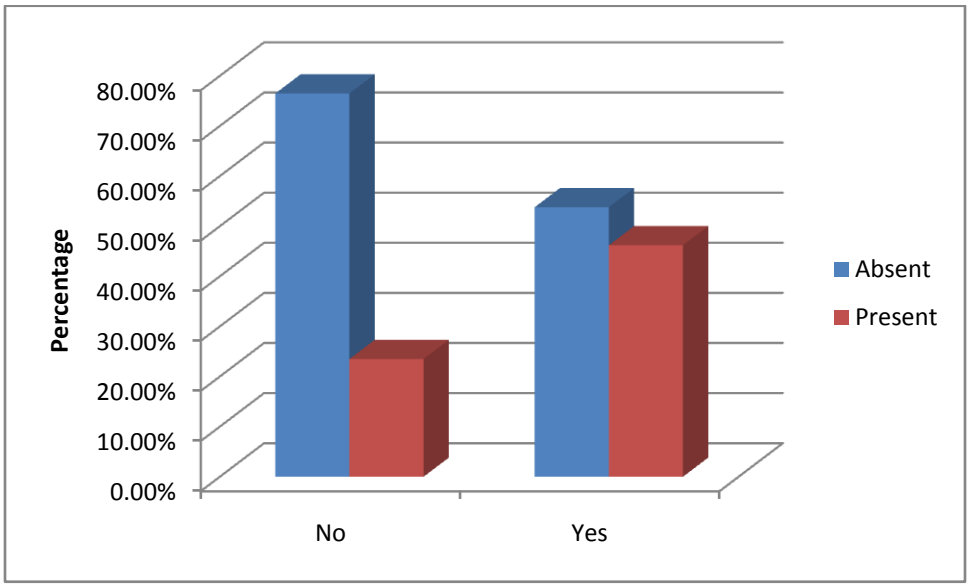

Figure 25- Bilateral phrenic nerve motor amplitude and latency involvement relation with respiratory involvement in LGBS patients

There were 8 cases who had completely inexcitable phrenic nerve amongst whom 3 cases showed respiratory involvement.

COMPLETE NCV ANALYSIS

RELATION WITH MOTOR NERVES' DISTAL LATENCY

When median motor distal latencies were studied, on both sides latency was found to be abnormal (increased or no response) in 64.3% (9 out of 14) cases with respiratory involvement (p-value 0.170) and 42.4% (14 out of 33) cases without respiratory involvement, i.e. latency was found to be abnormal for median nerve more in those with respiratory involvement. Five patients showed completely no response on both sides and among them 2 cases showed respiratory involvement.

Ulnar nerve distal latency was increased or showed no response on left side in 50% (7 out of 14) cases with respiratory involvement (p-value 0.501) as compared to 39.4% (13 out of 33) without respiratory involvement. Similarly on right side, abnormality was seen more in respiratory involvement group 57.1% (8 out of 14) (p-value 0.188) as compared to 36.4% (12 out of 33) in cases without respiratory involvement. Seven cases had completely inexcitable ulnar motor nerves with 4 of them having respiratory involvement.

Not much difference was noted in peroneal motor distal latencies on both sides. Cases with respiratory involvement having abnormal (increased latency or no response) values was observed in 64.3% (9 out of 14) on left side (p-value 0.966) and in 57.1% (8 out of 14) on right side (p-value 0.978) when compared with those without respiratory involvement having abnormal values in 63.6% (21 out of 33) on left and 57.6% (19 out of 33) on right side. Ten cases showed completely no response with 5 of them having respiratory involvement.

Tibial motor distal latencies were abnormal (increased or no response) more in respiratory involvement group with 57.1% (8 out of 14) on left (p-value 0.464) and 57.1% (8 out of 14) on right side (p-value 0.355) as compared to abnormal values in 45.5% (15 out of 33) on left and 42.4% (14 out of 33) on right side without respiratory involvement. Ten cases had completely no response and 6 of them progressed to respiratory difficulty.

There were 3 cases in which all four motor nerves were completely inexcitable and 2 of them progressed to respiratory difficulty. These 2 cases also had completely inexcitable phrenic nerves.

Table 28- Peripheral motor nerves' distal latency co-relation with respiratory involvement in LGBS								
Distal latency		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Median motor left	Abnormal	14	42.4%	9	64.3%	23	1.880	0.170
	Normal	19	57.6%	5	35.7%	24		
Median motor right	Abnormal	14	42.4%	9	64.3%	23	1.880	0.170
	Normal	19	57.6%	5	35.7%	24		
Ulnar motor left	Abnormal	13	39.4%	7	50.0%	20	0.452	0.501
	Normal	20	60.6%	7	50.0%	27		
Ulnar motor right	Abnormal	12	36.4%	8	57.1%	20	1.736	0.188
	Normal	21	63.6%	6	42.9%	27		
Peroneal motor left	Abnormal	21	63.6%	9	64.3%	30	0.002	0.966
	Normal	12	36.4%	5	35.7%	17		
Peroneal motor right	Abnormal	19	57.6%	8	57.1%	27	0.001	0.978
	Normal	14	42.4%	6	42.9%	20		
Tibial motor left	Abnormal	15	45.5%	8	57.1%	23	0.537	0.464
	Normal	18	54.5%	6	42.9%	24		
Tibial motor right	Abnormal	14	42.4%	8	57.1%	22	0.855	0.355
	Normal	19	57.6%	6	42.9%	25		

Figure 26- Relation of peripheral motor nerve latency findings with respiratory involvement in LGBS patients

RELATION WITH MOTOR NERVES' AMPLITUDE

When median motor amplitude was studied, amplitude was found to be abnormal (decreased or no response) in 71.4% (10 out of 14) cases with respiratory involvement on left side (p-value 0.372) and 64.3% (9 out of 14) on right side (p-value 0.812) and 57.6% (19 out of 33) cases without respiratory involvement on left side and 60.6% (20 out of 33) on right side, i.e. amplitude was found to be abnormal (decreased/ no response) for median nerve more in those with respiratory involvement. Five patients showed completely no response on both sides and among them 2 cases showed respiratory involvement.

Ulnar nerve amplitude was decreased or showed no response on left and right side in 78.6% (11 out of 14) cases with respiratory involvement as compared to 63.6% (21 out of 33) without respiratory involvement, thus suggesting abnormal finding which might predict respiratory failure but did not reach significant p-value (p-value 0.315) in view of small sample size of this study and therefore needs further evaluation with larger sample

size. Seven cases had completely inexcitable ulnar motor nerves and 4 of them had respiratory involvement.

Minor difference was noted for peroneal motor amplitude on both sides, with cases with respiratory involvement having abnormal (decreased or no response) values in 64.3% (9 out of 14) and in 57.6% (19 out of 33) when compared with those without respiratory involvement (p-value 0.668). Ten cases showed completely no response and 5 of them had respiratory involvement.

Tibial motor amplitude was abnormal (decreased or no response) in 57.1% (8 out of 14) on left (p-value 0.675) and 50% (7 out of 14) on right side (p-value 0.384) in patients with respiratory involvement as compared to abnormal values in 63.6% (21 out of 33) on left and 63.6% (21 out of 33) on right side without respiratory involvement. Ten cases had completely no response with 6 of them progressing to respiratory difficulty.

Table 29- Peripheral motor nerves' amplitude co-relation with respiratory involvement in LGBS								
Amplitude		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Median motor left	Abnormal	19	57.6%	10	71.4%	29	0.798	0.372
	Normal	14	42.4%	4	28.6%	18		
Median motor right	Abnormal	20	60.6%	9	64.3%	29	0.056	0.812
	Normal	13	39.4%	5	35.7%	18		
Ulnar motor left	Abnormal	21	63.6%	11	78.6%	32	1.009	0.315
	Normal	12	36.4%	3	21.4%	15		
Ulnar motor right	Abnormal	21	63.6%	11	78.6%	32	1.009	0.315
	Normal	12	36.4%	3	21.4%	15		
Peroneal motor left	Abnormal	19	57.6%	9	64.3%	28	0.184	0.668
	Normal	14	42.4%	5	35.7%	19		
Peroneal motor right	Abnormal	19	57.6%	9	64.3%	28	0.184	0.668
	Normal	14	42.4%	5	35.7%	19		
Tibial motor left	Abnormal	21	63.6%	8	57.1%	29	0.175	0.675
	Normal	12	36.4%	6	42.9%	18		
Tibial motor right	Abnormal	21	63.6%	7	50.0%	28	0.759	0.384
	Normal	12	36.4%	7	50.0%	19		

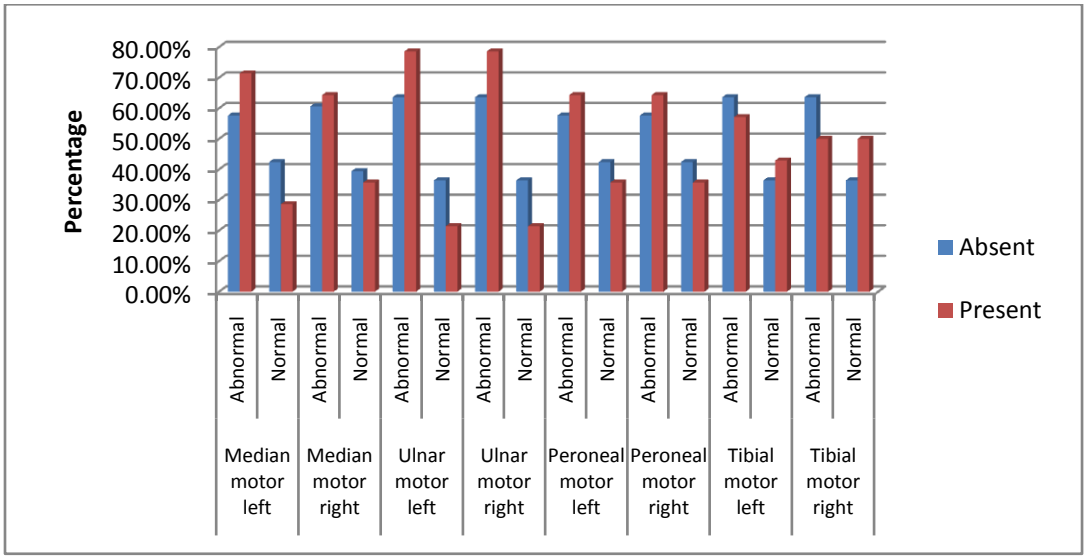

Figure 27- Relation of peripheral motor nerve amplitude findings with respiratory involvement in LGBS patients

RELATION WITH MOTOR NERVES' VELOCITY

Median motor velocities were more commonly found to be abnormal (decreased/ no response) in patients with respiratory involvement on both sides. On left side 64.3% (9 out of 14) and on right side 57.1% (8 out of 14) on right side had abnormal median motor conduction velocities along with respiratory involvement as compared to those without respiratory involvement having abnormal velocities in 27.3% (9 out of 33) on left and 30.3% (10 out of 33) on right side. Although giving a significant p- value only for left side velocities (p-value 0.024) (attributable to small sample size of this study) with patients having abnormal median motor velocities being at 4.8 times greater risk of having respiratory involvement.

Median nerve velocities were not recorded in 6 patients (no response noted) and 2 had respiratory involvement.

Ulnar nerve velocities did not show any significant abnormalities for predicting respiratory involvement in LGBS.

Peroneal nerve velocities can be used to predict respiratory involvement in LGBS as trend of p-value was towards significance atleast for left side (p-value 0.055). Decrease in velocity or no response was seen in 78.6% (11 out of 14) on both sides in those with respiratory involvement as compared to those who did not progress to respiratory difficulty. Ten cases showed completely no response and 5 of them had respiratory involvement.

Tibial nerve velocities showed a variable response on both sides. Where on left side only 64.3% (9 out of 14) had decreased tibial nerve velocity or no response with respiratory involvement (p-value 0.716) as compared to 69.7% (23 out of 33) without respiratory involvement, on right side 85.7% (12 out of 14) had abnormal velocities with respiratory involvement (p-value 0.047) as compared to 51.5% (17 out of 33) without respiratory involvement. It can be said that those patients with decreased tibial nerve velocities or no response are 5.65 times at increased risk of progression to respiratory failure atleast on right side. Ten cases had completely no response and 6 of them progressed to respiratory difficulty. Three out of these 10 had no response in phrenic nerve also with 2 of them having respiratory compromise.

Table 30- Peripheral motor nerves' velocity co-relation with respiratory involvement in LGBS									
Velocity		Respiratory involvement				Total	Chi-square value	p-value	odd ratio (95%CI)
		Absent		Present					
Median motor left	Abnormal	9	27.3%	9	64.3%	18	5.699	0.024	4.8(1.26-18.23)
	Normal	24	72.7%	5	35.7%	29			
Median motor right	Abnormal	10	30.3%	8	57.1%	18	2.997	0.108	
	Normal	23	69.7%	6	42.9%	29			
Ulnar motor left	Abnormal	8	24.2%	4	28.6%	12	0.097	0.756	
	Normal	25	75.8%	10	71.4%	35			
Ulnar motor right	Abnormal	7	21.2%	5	35.7%	12	1.087	0.297	
	Normal	26	78.8%	9	64.3%	35			
Peroneal motor left	Abnormal	15	45.5%	11	78.6%	26	4.362	0.055	
	Normal	18	54.5%	3	21.4%	21			
Peroneal motor right	Abnormal	16	48.5%	11	78.6%	27	3.640	0.105	
	Normal	17	51.5%	3	21.4%	20			
Tibial motor left	Abnormal	23	69.7%	9	64.3%	32	0.132	0.716	
	Normal	10	30.3%	5	35.7%	15			
Tibial motor right	Abnormal	17	51.5%	12	85.7%	29	4.865	0.047	5.65(1.09-29.26)
	Normal	16	48.5%	2	14.3%	18			

Figure 28- Relation of peripheral motor nerve velocity findings with respiratory involvement in LGBS patients

Unilateral or bilateral involvement of peripheral motor nerves as increased latency or decreased amplitude or decreased velocities or no response at all had no significant co-relation in predicting respiratory involvement in LGBS patients in our study. This finding was limited because of the small sample size of our study and requires further corroboration with larger studies.

Overall, there were 3 patients who had completely inexcitable all 4 motor nerves along with phrenic nerve and 2 of such patients had respiratory involvement.

F-wave latency and F- wave persistence were also analysed for all four peripheral motor nerves but no significant co- relation was found with prediction of respiratory involvement in LGBS patients.

Sensory nerve conduction studies had no significant co-relation with respiratory involvement in LGBS patients (p-value >0.05).

NCV findings of GBS patients were also classified into axonal and demyelinating varieties and its co-relation was studied to predict respiratory involvement but was insignificant (p-value 0.558).

It was observed that amongst patients with respiratory involvement 4 cases (28.6%) were axonal type and 10 cases (71.4%) were demyelinating type. For one patient the variety of GBS could not be assessed in view of completely no response of all nerves on NCV studies.

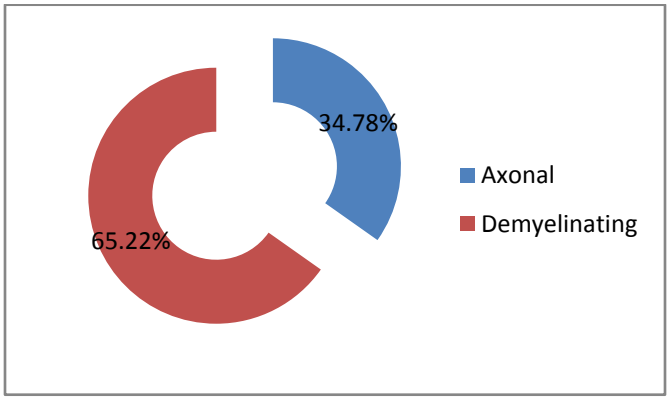

Figure 29- Frequency of LGBS abnormality types

Table 31- LGBS variety and relation with respiratory involvement

		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Type of variety	Axonal	12	37.5%	4	28.6%	16	0.342	0.558
	Demyelinating	20	62.5%	10	71.4%	30		
Total		32	100.0%	14	100.0%	46		

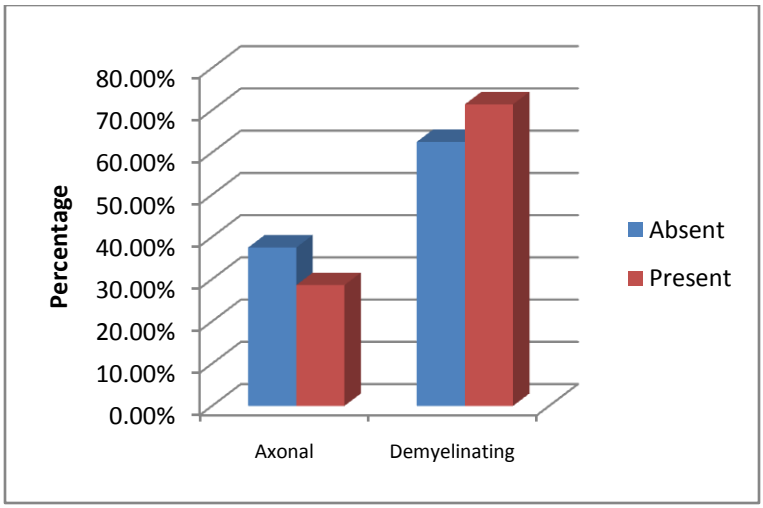

Figure 30- Type of LGBS variety and relation with respiratory involvement

RELATION WITH CRANIAL NERVES

7 cases (14.9%) showed cranial nerve involvement in the form of unilateral or bilateral facial nerve palsy with 57.1 % (4 of them) showing respiratory involvement but found to have insignificant co-relation (p- value 0.173). There was a single case in our study which along with facial nerve palsy also showed bulbar paralysis and had respiratory involvement as well.

Table 32- Cranial nerve involvement in LGBS

Cranial nerve involvement	No. of cases	Percentage
No	40	85.1%
Yes	7	14.9%
Total	47	100.0%

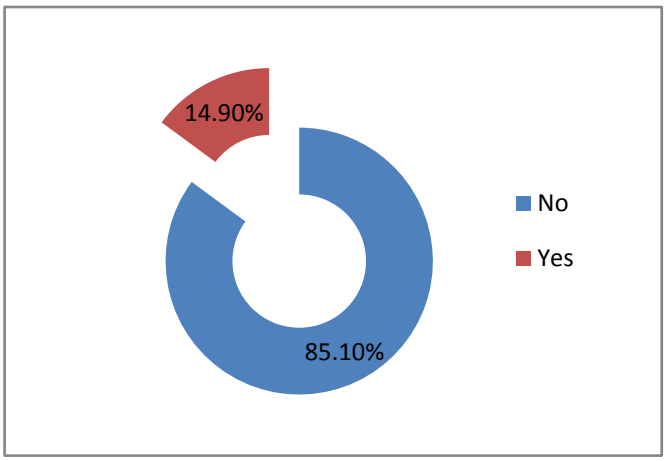

Figure 31- Cranial nerve involvement in LGBS

Table 33- Cranial nerve involvement role in predicting respiratory involvement in LGBS patients								
		Respiratory involvement				Total	Chi-square value	p-value
		Absent		Present				
Cranial nerve involvement	No	30	75.0%	10	25.0%	40	2.943	0.173
	Yes	3	42.9%	4	57.1%	7		
Total		33	70.2%	14	29.8%	47		

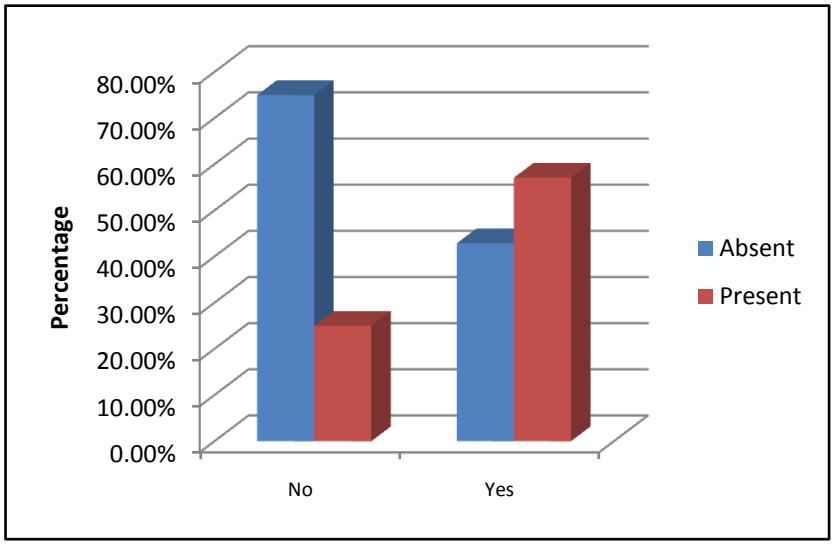

Figure 32- Relation of cranial nerve involvement with respiratory involvement in LGBS patients

DISCUSSION

Guillain Barre Syndrome (GBS) is an acute autoimmune polyradiculoneuropathy associated with significant morbidity. During hospitalization, some of the patients of GBS are intubated for respiratory failure and their course can be further complicated by dysautonomia and various intensive care units (ICU) related complications [43]. The outcome of GBS has varied widely in published series with mortality rates ranging between 1-18% [70] and remaining higher (12-20%) in those who required mechanical ventilation [6]. The mortality in ventilated patients was higher (20%) in the study by Lawn *et al*. [43] and 38.3% in the series by Taly *et al* [50].

Uma Sundar et al. [71] in their study mentioned that neuromuscular respiratory failure is one of the major factors influencing morbidity and mortality in GBS. In another study [72], it was found that respiratory failure in GBS results from a combination of factors- tongue, pharyngeal and laryngeal weakness causing poor clearing of oral secretions and diaphragmatic intercostals muscle weakness causing progressive type 2 respiratory failure, leading to atelectasis and progressive hypoxia.

To overcome these complications and to decrease the morbidity and mortality associated with LGBS, with the available studies this study was planned to assess phrenic nerve conduction abnormalities in LGBS, type of abnormalities that can be observed and if they can be used to predict the onset of respiratory failure in LGBS for early intervention.

In our study, total 47 patients were enrolled over a period of 18 months who were 18 or more than 18 years of age and presented with clinical suspicion of LGBS. During hospital stay, they were followed up for development of any respiratory distress. Among total of 47 cases, 29.8% (14 patients) had respiratory involvement with 64.3% (9 patients) requiring

mechanical ventilation. Various clinical and NCV parameters along with phrenic nerve conduction studies were compared with patients having respiratory involvement and those patients without respiratory involvement. We found that 44.7% (21 patients) patients were 31 to 50 years of age, 25.5% (12 patients) were less than 30 years of age and 29.8% (14 patients) were more than 50 years of age. Fifty percent patients with respiratory involvement were 31 to 50 years of age. Mean age of patients who had respiratory involvement was 39.57 ± 16.20 years. No significant co-relation of respiratory involvement in LGBS patients with age was seen (p-values >0.05). These findings were in consistence with previous studies done by Islam et al. [73], Walgaard C and Lawn ND [25, 23], stating age was not associated with mechanical ventilation among patients with GBS. Even according to study done by Birinder S Paul et al. [60], there was no statistically significant difference between the two groups (ventilated and non- ventilated) in age and presence of any associated co-morbidity.

In our study, 21 patients were females and 26 patients were males. Amongst 14 patients with respiratory involvement, 50% were males and other 50% were females suggestive that gender has no co- relation with respiratory involvement in LGBS (p-value 0.633). In a study by Maher Bano [74], a total of 110 patients were included. Out of these 110 patients, 76 (69.09%) patients were male and 34 (30.91%) patients were females. 24 male patients had respiratory distress while 52 patients had no respiratory distress and 9 female patients had respiratory distress while 25 patients had no respiratory distress with insignificant p value of 0.589 while there was no significant age predilection also. Uma Sundar et al. [71] in their study compared data in ventilated and non ventilated groups and found that age, gender, preceding infection, bifacial or neck weakness and early upper limb paralysis did not predict the requirement for mechanical ventilation. Similar findings were seen in study done by Barun Kumar Sen

and Alak Pandit [45] stating that age, gender, preceding infection and type of GBS did not influence the development of respiratory failure ($P > 0.05$).

Clinical assessment of all patients with suspicion of LGBS was done.
In our study, the mean respiratory rate for patients without respiratory involvement was 20± 2.29 per minute whereas for patients with respiratory involvement it was 26.46± 2.18 per minute which was more in this group and it can be anticipated that more the respiratory rate on admission, more are the chances of patient progressing to respiratory distress. Then single breath count was noted for each patient on admission. In our, study mean SBC was found to be 23.22 ± 11.141. Patients with respiratory distress had a mean SBC of 16.38 ± 7.35 and those without respiratory involvement had a mean of 25.91 ± 11.31, giving a p-value of 0.008 suggestive of significant co-relation with SBC and respiratory involvement prediction in LGBS patients. This in accordance to the study by Barun kumar Sen and Alak pandit [45], where mean single breath count was 26.7 ± 10.35 (minimum- 10, maximum- 46) and those who developed respiratory failure it was 19 ± 9.21, whereas it was 29.95 ± 9.07 in those who did not develop respiratory failure, i.e., significantly lower in those who developed respiratory failure (p-value 0.000022). They found that SBC is a simple bedside test and is significantly low in those patients who developed respiratory failure.

Then power was assessed for all patients and power grading was compared in both groups with and without respiratory involvement. In our study we found that in all muscle groups power less than equal to 3 and handgrip less than 50% was seen more commonly in group which progressed to respiratory failure as compared to non-ventilated group, although only power grade less than equal to 3 in left shoulder and elbow and left ankle reached a significant p-value. Therefore, it can be concluded that lesser the power grade, more the limb weakness, more severe the disease and more the chances of patient progressing to

respiratory failure. Even according to Birinder S Paul's study [60], they summarised that presence of upper respiratory infection as one of the antecedent events, neck and bulbar weakness (P<0.001), simultaneous onset of motor weakness in UL and LL as the initial symptom (P<0.001) with upper limb power< 3 at nadir (P<0.001), shorter duration from onset to bulbar weakness and confinement to bed (P=0.001), and bilateral facial involvement (P<0.01) were more frequently associated with the need for ventilation. According to Cameron Green et al [75] also, simultaneous onset of weakness in upper and lower limbs, upper limbs' power grade < 3/5 and bulbar involvement should be taken as warning signs for predicting respiratory involvement in LGBS patients. According to numerous studies, muscle weakness was significantly greater at hospital admission in patients who later required mechanical ventilation [25, 76-80]. Rajesh Verma et al. [79] also did a study saying that presence of autonomic dysfunction, bulbar, neck flexor and severe limb weakness along with conduction block on neurophysiological assessment increased the need for mechanical ventilation. As quoted in a study by Lawn ND et al. [23], no significant differences were noted between those patients who received ventilation and those who did not for age, sex and for the presence of any preceding gastrointestinal illness, upper limb paralysis and pulmonary disease. Although upper limb paralysis was identified in a higher proportion of patients who subsequently received mechanical ventilation, but this did not reach statistical significance. Patient dependent factors such as age and the presence of preceding chronic pulmonary disease did not predict the progression to mechanical ventilation.

In our study, for all clinically suspected cases of LGBS, provisional diagnosis of LGBS was kept and NCV studies were performed. Patients whose NCV studies showed LGBS, their phrenic nerve conduction studies were then performed. Phrenic nerve was involved either unilaterally or bilaterally as amplitude or latency abnormality alone or both in 87.2 % patients (41 patients). Amongst 33 patients without respiratory

involvement, phrenic nerve conduction was abnormal in 29 patients (87.9%). Abnormalities found in LGBS patients in phrenic nerve conduction studies were decrease in amplitude, increase in latency and few patients showing no response at all. Decrease in amplitude was a more common abnormality than increase in latency. Decrease in amplitude or increase in latency or no response for phrenic nerve showed no significance in predicting respiratory involvement in LGBS according to our study.

Left phrenic nerve amplitude was found to be decreased (23 cases- 48.9%) or showing no response (14 cases- 29.8%) in 78.7 % cases (37 patients) amongst whom 29.7% (11 patients) showed respiratory involvement. Right phrenic nerve amplitude was found to be decreased (29 cases- 61.7%) or showing no response (9 cases- 19.1%) in 80.8 % cases (38 patients) amongst whom 31.6 % (12 patients) showed respiratory involvement. Bilateral involvement of phrenic nerve amplitude that is decreased amplitude or no response was seen in 78.7% cases (37 cases) with 29.7% (11 cases) having respiratory involvement.

On left phrenic nerve conduction studies, 38.3% cases (18 cases) showed increase in latency (10 cases- 21.3%) or no response at all (8 cases- 17%) , amongst whom 48.9 % (7 cases) had respiratory involvement. On right side, 49 % cases (23 cases) showed increase in latency (10 cases- 21.3%) or no response at all (13 cases- 27.7%) amongst whom 39.1 % (9 cases) had respiratory involvement. Bilateral involvement of phrenic nerve with increase in latency or no response at all was seen in 34% cases (16 cases) amongst whom 43.8 % (7 cases) showed respiratory involvement. All phrenic nerve abnormalities' co-relation with respiratory involvement in LGBS gave insignificant p- value of >0.05. According to our study, no significant co-relation was found with unilateral or bilateral abnormality of phrenic nerve either as decreased amplitude or as increased latency or no response at all with respiratory involvement in LGBS patients.

Simultaneous bilateral involvement of phrenic nerve amplitude and latency was also compared. It was observed in 27.7% (13 cases) of total cases and respiratory involvement was seen in 46.2 % cases (6 cases out of these 13 cases) but was found to be insignificant co-relation with p- value of 0.129.

According to an abstract submitted by Michael Rubin [81], respiratory embarrassment was not found to correlate with the presence of bulbar palsy, anti-ganglioside antibodies, or the results of conventional peripheral nerve conduction studies, including the tibial, peroneal, median, or ulnar nerves. Respiratory failure in GBS may be predicted by the presence of increased summed latency or motor amplitude of bilateral phrenic nerves although larger studies are warranted to confirm this notion.

In a study by U Zifko et al. [82], the incidence of phrenic nerve involvement and the predictive value of phrenic nerve conduction and diaphragmatic needle EMG were studied in 40 patients with Guillain- Barre syndrome within the first three days of admission to hospital. The negative peak onset latency of the diaphragmatic compound muscle action potential (CMAP), and its amplitude, duration, and area were abnormal in 83% (our study having phrenic nerve involvement in 87.2% patients). The need for ventilation was correlated with diaphragmatic CMAP amplitude (P = 0.005), and area (P = 0.001), but not with latency or duration. Abnormalities in diaphragmatic needle EMG were found in 45%, mainly a decreased number of motor unit potentials. The abnormalities correlated with the need for ventilation (P = 0.013). Of the 40% who required ventilation, all had either abnormal phrenic conduction or abnormal diaphragmatic needle EMG, or both. Patients who required mechanical ventilation had significantly lower diaphragmatic CMAP amplitude, smaller negative peak area and lower thenar CMAP than those who did not require ventilation. No relation was found between phrenic nerve latency, duration of the diaphragmatic CMAP, distal median motor nerve latency, median motor nerve conduction velocity and the need for ventilation.

Abnormal diaphragmatic EMG findings correlated with the need for mechanical ventilation. All 16 patients who required mechanical ventilation showed either abnormal phrenic nerve conduction (14, 88%), abnormal diaphragmatic EMG (11, 69%), or both abnormalities (9, 56%). Three of the 16 ventilated patients had normal median nerve conduction studies. Among the 24 patients who did not require ventilation, 19 (79%) had abnormal phrenic nerve conduction studies and seven (29%) also had abnormal diaphragmatic EMG. The five patients with normal respiratory electrophysiological studies did not require ventilation. Forty two percent of patients with abnormal phrenic nerve latency, 82% with diminished diaphragmatic CMAP amplitude and 78% with abnormal diaphragm EMG required mechanical ventilation. Their findings indicate that involvement of the phrenic nerve and diaphragm is common at an early stage of the disease, occurring in 88% of patients. Furthermore, some respiratory electrophysiological variables are sensitive predictors of severe respiratory failure, and are superior to spirometry in terms of early recognition of patients who are at risk of severe diaphragmatic weakness. Prolonged phrenic nerve latency was the most common finding (78%) (as compared to phrenc nerve amplitude abnormalities being more common in our study), being present in ventilated as well as in non-ventilated patients which suggested that either axonal degeneration or mild segmental demyelination is occurring in the early stages of Guillain Barre Syndrome, although the prolongation was only mild and did not correlate with the FVC or with the need for ventilation. However, features other than the prolonged latency proved important. The diaphragmatic CMAP amplitude and negative peak area were both reduced, but the duration was unchanged. This suggested that either axonal degeneration or demyelination caused a pure conduction block in phrenic nerve fibres distal to the point of stimulation in the neck. Prolongation of the duration of the diaphragmatic CMAP would have indicated disproportionate slowing of conduction in nerve fibres that were not blocked. This plus the only mildly

prolonged latency, suggest that phase cancellation was unlikely to have been an important mechanism of CMAP amplitude and area reduction [83]. They also found that although phrenic nerve conduction and needle EMG of the diaphragm showed a sensitivity of 100% in predicting respiratory failure, they were not specific. Patients without respiratory failure had abnormal phrenic nerve latencies (79%), abnormal diaphragmatic CMAP amplitudes (29%) and abnormal findings on needle EMG of the diaphragm (29%). However, no patient with normal electrophysiological respiratory studies developed respiratory failure. Thus patients with Guillain Barre Syndrome and normal phrenic nerve conduction and normal diaphragmatic needle EMG, are unlikely to develop significant ventilatory failure. The mean distal CMAP amplitude of limb nerves is reported to be a good predictor of outcome in Guillain Barre Syndrome [36, 42, 84]. Therefore, U Zifko et al. [82] concluded that phrenic nerve conduction studies and diaphragmatic needle EMG be performed in the early stage of Guillain-Barre syndrome to detect patients who have respiratory muscle involvement and are at risk of respiratory decompensation.

In our study, there were 6 patients with completely normal phrenic nerve bilaterally and 2 of them showed respiratory involvement with one of them even requiring mechanical ventilation. These two patients had normal phrenic nerve conduction and also not much motor nerve conduction abnormalities were noted but respiratory involvement was seen. Mainly F-wave abnormalities were seen in these two patients and according to a study done by Uma Sundar et al. [71], commonest finding in ventilated as well as non-ventilated groups was radiculopathy, as evidenced by impaired H reflex and F waves in the motor nerves. Also according to U Zifko et al. [82], of the 40% patients who required ventilation in their study, all had either abnormal phrenic conduction or abnormal diaphragmatic needle EMG, or both and so may be these two exceptional cases in our study might have had abnormal diaphragmatic needle EMG (which was not taken into consideration in our study) despite of normal phrenic nerve

conduction studies. This can also suggest that phrenic nerve conduction studies cannot solely predict respiratory compromise in GBS patients and can be further corroborated with needle EMG of diaphragmatic muscle. Even in our study there were 8 cases who had completely inexcitable phrenic nerve amongst whom 3 cases showed respiratory involvement and 5 cases did not.

As also stated by Durand MC et al. [78], phrenic nerve electrophysiology was not helpful for anticipating respiratory failure related to the disease, although this finding is controversial according to some other studies [54, 82].

In our study, the four peripheral motor nerves were also analyzed along with phrenic nerve- median, ulnar, peroneal and tibial nerves- for comparing if they could predict respiratory involvement in LGBS patients. Their latencies, amplitudes and velocities were evaluated. It was observed that latencies were prolonged, amplitudes were decreased and velocities slowed down on admission and they might predict respiratory failure although none of the values reached significance in view of small sample size of this study except for left median motor and right tibial motor velocities, although study done by U Zifco et al. [82] showed no relation of median motor nerve velocity with respiratory involvement in LGBS. In our study unilateral or bilateral involvement of peripheral motor nerves as increased latency or decreased amplitude or decreased velocities or no response at all had no significant co-relation in predicting respiratory involvement in LGBS patients. This finding was limited because of the small sample size of our study. So, further studies are needed to derive such conclusions on a larger scale.

Overall, there were 3 patients who had completely inexcitable all 4 motor nerves along with phrenic nerve and 2 of such patients had respiratory involvement.

In study by Uma Sundar et al. [71], electrophysiological studies done prior to maximum disability were available. Marked reduction in CMAP amplitude in more than one motor nerve tested was seen in 6/11 ventilated patients and in 2/13 non-ventilated patients. In the ventilated group, two patients had two or more inexcitable nerves. Temporal dispersion (4/11 ventilated vs. 4/13 nonventilated), conduction block (1/11 ventilated vs. 3/13 non-ventilated) and significant motor nerve conduction velocity slowing (3/11 ventilated vs. 5/13 non-ventilated) were infrequent in both groups (although median motor nerve velocities showed significant co-relation on left side in our study which can be attributed to small sample size of this study and it was a unilateral finding and needs further evaluation on larger scale). Denervation changes on the EMG in more than one muscle tested were seen in 5/11 ventilated patients. Sensory nerve action potentials (either upper limb or lower limb) were abnormal in 7/11 ventilated and 8/13 non-ventilated patients. Prolongation of distal motor latency in two or more nerves was seen in 5/11 ventilated and 6/13 non-ventilated patients. Markedly low CMAP amplitudes seen in 6/11 ventilated patients could represent either severe distal demyelination or axon loss. Only 2/13 patients in the non-ventilated group had this finding. Evaluation of motor nerves by quantitation of CMAP amplitudes is one of the most important early studies in GBS. Low CMAP amplitude on distal stimulation, which could indicate either distal demyelination or axonopathy, was overall a much commoner finding (14/24) than temporal dispersion or conduction block, each of which indicates more proximal segmental demyelination. Three studies have shown that a markedly low distal CMAP indicates poor prognosis in GBS. Cornblath et al. [42] from pooled data related to the North American Guillain Barre Plasmapheresis trial, found a CMAP amplitude less than 20% of lower limit of normal, to be associated with a poor prognosis. Miller et al [85] studied 65 GBS patients at a mean of 18 days after onset of weakness and found poor prognosis to be related to CMAP amplitude less than 10% of lower limit of normal.

Feasby et al [86] studied the median, ulnar and peroneal nerves of 25 patients with GBS prospectively and concluded that CMAP amplitudes of 10% of control mean for at least one nerve had a poorer outcome. They also said that the presence of inexcitable motor nerves is associated with a poor outcome (Feasby 1986; Miller 1982, 1988) [86, 85]. In our study, only categorical evaluation was done to compare respiratory involvement with those without respiratory involvement for peripheral nerve studies. In the series by Brown WF et al [87], six of 11 ventilated patients and two of 13 non-ventilated patients, had markedly attenuated CMAP amplitudes in more than one motor nerve. In addition, in the ventilated group, two patients had more than one inexcitable motor nerve, this finding being absent in the non-ventilated group. Among the five patients in the ventilated group with prolonged distal latency, three also had markedly low CMAP amplitude. Low CMAP amplitude on distal stimulation is not necessarily a sign of axonopathy, as distal demyelination could give a similar finding. However, the two patients with 'inexcitable nerves' and the five patients with denervation on EMG, all belonged to the group of six patients with 'low CMAP amplitudes', in the ventilated group, making a case for axonopathic GBS in these patients stronger. The presence of demyelination and axonopathy together in GBS has been well documented by Brown et al [87].

In our study, F-wave latency and F- wave persistence were also analysed for all four motor nerves but no significant co-relation was found with prediction of respiratory involvement in LGBS patients. Even sensory involvement along with sensory nerve electrodiagnostic studies done on median, ulnar and sural nerves showed no significant co-relation with respiratory involvement in LGBS.

On the basis of NCV studies, patients were categorised to have axonal and demyelinating varieties of LGBS and their co-relation observed with respiratory involvement in LGBS patients but gave insignificant findings. Cranial nerve involvement might also predict respiratory involvement in

LGBS patients but our study was inconclusive for relation between respiratory compromise and cranial nerve deficits in LGBS patients although cranial nerve involvement mostly seventh nerve palsy either unilateral or bilateral was seen in 7 patients with 4 of them progressing to respiratory involvement. Although in a study by Nicholas D. Lawn et al. [23], it was observed that presence of bilateral facial palsy, autonomic dysfunction and rapid disease progression were also associated with increased risk of intubation. Even according to Birinder S Paul et al. [60], bilateral facial nerve involvement was observed more frequently in the patients within ventilated group (P<0.001). The clinical and electrophysiological factors associated with need for mechanical ventilation in study by Rajesh Verma et al. [79] included presence of autonomic dysfunction, cranial nerve palsy including facial palsy and bulbar palsy, neck flexor weakness, low MRC sum score (≤30) on admission and low proximal CPN CMAP amplitude to distal CPN CMAP amplitude ratio. However, on multivariate analysis, none of them was significant. Even our study showed no co-relation of cranial nerve involvement with respiratory deterioration in LGBS patients. They concluded that GBS is an immune mediated disorder affecting predominantly the young population usually preceded by infection in one third of the cases. Axonal variety was more common. Factors like autonomic dysfunction, poor muscle power grade, need for mechanical ventilation and axonal pattern on electrophysiology were associated with poor functional outcome. Although our study did not give any co-relation with cranial nerve involvement and LGBS variety in view of small sample size of our study and demyelinating variety was found to be more common in both groups with and without respiratory involvement in our study.

SUMMARY AND CONCLUSION

1. Of total of 47 cases enrolled in our study, 29.8 % (14 patients) had respiratory involvement with 64.3% (9 out of these patients) requiring mechanical ventilation.
2. Age and gender did not show significant co-relation in predicting respiratory involvement in LGBS patients.
3. Patients with increased respiratory rate on admission have increased chances of progressing to respiratory involvement in LGBS patients.
4. Lower single breath count (SBC) (mean SBC 16.38±7.35) suggested increased severity of the disease and more chances of having respiratory involvement.
5. Increased severity of limb weakness (power grade ≤ 3) on admission suggested increased disease severity and co-related with increased risk of progressing to respiratory involvement.
6. Phrenic nerve conduction abnormalities seen in LGBS patients in our study were decreased amplitude or increased latency or both or no response at all.
7. Phrenic nerve conduction studies alone either unilaterally or bilaterally cannot predict respiratory involvement in LGBS patients. Small sample size of our study was a limitation for this conclusion and requires further analysis on a large scale.
8. According to our study, peripheral motor nerve conduction studies mainly have diagnostic importance in LGBS and unilateral or bilateral studies might help predict respiratory involvement showing decreased amplitudes, increased latencies and decreased velocities but again requires detailed assessment with a large sample size.
9. F-wave latency and persistence and sensory nerve conduction studies were not predictive for respiratory compromise in LGBS patients.
10. Most common cranial nerve abnormality observed in our study was seventh nerve palsy.

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KEY TO MASTER CHART

MRD	Medical Record Department
U/L	Unilateral
B/L	Bilateral
NCV	Nerve Conduction Velocity
DMCH	Dayanand Medical College And Hospital
IVIg	Intravenous Immuneglobulin
I/V	Intravenous
DOR	Discharge On Request
DAMA	Discharge Against Medical Advice
OPD	Outpatient Department

S.NO	NAME	MRD NO.	AGE (YEARS)	SEX	RESPIRATORY RATE (PER MINUTE) (on admission)	SINGLE BREATH COUNT	POWER (ON ADMISSION)												BILATERAL DEEP TENDON REFLEXES	BILATERAL SENSORY SYSTEM INVOLVEMENT (CLINICAL)	PHR						
							UPPER LIMB						LOWER LIMB								AMPLITUDE (>320MICROVOLT)						
							RIGHT			LEFT			RIGHT			LEFT					MOTOR						
							SHOULDER	ELBOW	HAND GRIP	SHOULDER	ELBOW	HAND GRIP	HIP	KNEE	ANKLE	HIP	KNEE	ANKLE			LEFT		RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LE
1	AK	620103	63	MALE	20	20	4	4	80%	4	4	80%	4	4	4	4	4	4	ABSENT	NO	66.1	DECREASED	114.5	DECREASED	NO	YES	6
2	BK	623416	70	FEMALE	22	16	2	2	40%	2	2	40%	2	2	2	2	2	2	ABSENT	NO	251.1	DECREASED	83.3	DECREASED	NO	YES	8.4
3	SS	638200	44	MALE	20	24	5	5	90%	5	5	90%	3	3	3	3	3	3	ABSENT	NO	388.8	NORMAL	320.8	NORMAL	NO	NO	4.1
4	SK	646844	34	FEMALE	24	20	4	4	80%	4	4	80%	0	0	0	0	0	0	ABSENT	NO	486.2	NORMAL	161	DECREASED	YES	NO	10.9
5	AK	660069	21	MALE	22	30	4+	4+	75%	4+	4+	75%	4-	4	4	4-	4	4	ABSENT	YES	NO RESPONSE	NO RESPONSE	121.1	DECREASED	NO	YES	NO RESPONSE
6	DS	663471	43	MALE	20	35	4+	4	30%	4+	4	30%	4+	4-	4-	4+	4-	4-	ABSENT	NO	NO RESPONSE	NO RESPONSE	47	DECREASED	NO	YES	NO RESPONSE
7	R	667314	33	FEMALE	22	26	3	4	40%	4	4	40%	3	4	4	3	4	4	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
8	HS	678065	28	MALE	22	19	0	0	10%	0	0	10%	0	0	0	0	0	0	ABSENT	NO	82.4	DECREASED	73.3	DECREASED	NO	YES	3.3
9	PG	677371	24	MALE	24	18	3	4-	20%	3	4-	20%	3	4-	4-	3	4-	4-	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
10	NK	679377	74	FEMALE	18	30	4	4	80%	4	4	80%	3	3	3	3	3	3	ABSENT	NO	112.1	DECREASED	133.3	DECREASED	NO	YES	8
11	MK	698042	34	FEMALE	26	11	2	2	30%	1	1	30%	0	0	0	1	1	1	ABSENT	NO	479.3	NORMAL	561.1	NORMAL	NO	NO	6.2
12	DK	702439	36	FEMALE	20	60	4-	4-	20%	4-	4-	20%	4-	3	1	3	3	1	ABSENT	NO	182.3	DECREASED	133.6	DECREASED	NO	YES	2
13	NG	712126	36	FEMALE	22	10	4	4	10%	4	4	10%	2	2	2	2	2	2	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
14	ES	715561	27	FEMALE	24	33	4	4	90%	4	4	90%	2	3	4	2	3	4	ABSENT	NO	191.6	DECREASED	214.1	DECREASED	NO	YES	7.8
15	TL	722336	46	MALE	20	22	4	4	80%	4	4	80%	4	4	4	4	4	4	ABSENT	NO	481.3	NORMAL	472.5	NORMAL	NO	NO	7.6
16	HK	726934	18	FEMALE	20	22	4	4	80%	4	4	80%	1	1	1	1	1	1	ABSENT	NO	400.3	NORMAL	489.4	NORMAL	NO	NO	4.4
17	RB	726888	32	FEMALE	22	30	4	4	60%	4	4	60%	4	4	4+	4	4	4+	ABSENT	NO	282.8	DECREASED	250.8	DECREASED	NO	YES	5.4
18	JK	727668	60	FEMALE	22	24	2	3	40%	2	3	40%	2	3	5	2	3	5	ABSENT	NO	163.4	DECREASED	193.8	DECREASED	NO	YES	5.6
19	SS	728987	50	MALE	20	18	4-	4+	60%	4-	4-	60%	4-	5	4-	4-	5	4-	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
20	S	734436	18	MALE	ON TRACHEOSTOMY	COULD NOT BE	1	1	30%	1	1	30%	1	1	1	1	1	1	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
21	SS	736396	65	MALE	22	20	1	1	20%	1	1	20%	2	1	2	2	1	2	ABSENT	NO	NO RESPONSE	NO RESPONSE	276.2	DECREASED	NO	YES	NO RESPONSE
22	GS	741216	24	MALE	22	32	3	2	40%	2	3	40%	2	3	3	2	3	3	ABSENT	NO	232.7	DECREASED	224	DECREASED	NO	YES	7
23	HS	765811	38	MALE	28	18	4	4	40%	4-	4-	30%	3-	4-	4-	4-	4	4	ABSENT	NO	100.2	DECREASED	167	DECREASED	NO	YES	6
24	BS	769025	54	MALE	30	4	1	1	20%	1	1	20%	1	1	1	1	1	1	ABSENT	YES	NO RESPONSE	NO RESPONSE	0.6	DECREASED	NO	YES	13.1
25	PCG	752729	69	MALE	26	17	1	1	30%	1	1	30%	1	1	1	1	1	1	ABSENT	NO	141.5	DECREASED	106.8	DECREASED	NO	YES	9.1
26	VR	783712	50	FEMALE	28	10	3	3	60%	2	2	60%	1	1	1	1	1	1	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
27	SK	803750	27	MALE	20	36	4	4	60%	4	4	60%	4	4	4	4	4	4	ABSENT	NO	162.3	DECREASED	145.8	DECREASED	NO	YES	1.2
28	AK	823722	50	FEMALE	26	10	4-	4-	80%	3	3	70%	4-	4-	4	3	3	3	ABSENT	NO	153.8	DECREASED	130.5	DECREASED	NO	YES	9.8
29	RK	979343	32	MALE	20	36	2	2	30%	2	2	30%	1	1	1	1	1	1	ABSENT	NO	110.7	DECREASED	86.7	DECREASED	NO	YES	3.8
30	RK	843997	39	FEMALE	16	26	2	2	40%	2	2	40%	2	2	2	2	2	2	ABSENT	NO	71	DECREASED	95.4	DECREASED	NO	YES	2.2
31	LD	848012	60	FEMALE	24	18	2	2	30%	2	2	30%	1	1	1	1	1	1	ABSENT	YES	85.4	DECREASED	82.8	DECREASED	NO	YES	9.6
32	MK	868747	45	FEMALE	22	26	3	3	60%	3	3	60%	3	3	3	3	3	3	ABSENT	NO	340.6	NORMAL	351.9	NORMAL	NO	NO	5.1
33	BDS	869851	50	MALE	28	24	3	3	40%	3	3	40%	1	1	1	1	1	1	ABSENT	NO	93.3	DECREASED	151.3	DECREASED	NO	YES	8.7

S.NO	NAME	MRD NO.	ENIC NERVE																							
			ONSET LATENCY (<8.0 MS)					INVOLVED		BILATERAL INVOLVEMENT OF BOTH AMPLITUDE AND LATENCY	MOTOR															VE
											ONSET LATENCY (<4.3 ms)						AMPLITUDE (>5.0 mV)								
			MOTOR								WRIST				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	WRIST				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT			
			FT	RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT	RIGHT		LEFT		RIGHT				LEFT		RIGHT				LEFT		
1	AK	620103	NORMAL	7.3	NORMAL	NO	NO	YES	YES	NO	3.2	NORMAL	3.3	NORMAL	NO	NO	3.6	DECREASED	3.3	DECREASED	NO	YES	64.8	NORMAL	57.5
2	BK	623416	INCREASED	4.8	NORMAL	YES	NO	YES	YES	NO	12.2	INCREASED		NO RESPONSE	NO	YES	0.2	DECREASED		NO RESPONSE	NO	YES	47.3	NORMAL	
3	SS	638200	NORMAL	3.8	NORMAL	NO	NO	NO	NO	NO	3.9	NORMAL	3.5	NORMAL	NO	NO	8.4	NORMAL	10.3	NORMAL	NO	NO	57.8	NORMAL	52.7
4	SK	646844	INCREASED	12.1	INCREASED	NO	YES	YES	YES	NO	11.8	INCREASED	12.5	INCREASED	NO	YES	0.9	DECREASED	0.9	DECREASED	NO	YES	33.1	DECREASED	30.9
5	AK	660069	NO RESPONSE	2.8	NORMAL	YES	NO	YES	YES	NO	5.6	INCREASED	5.1	INCREASED	NO	YES	4.7	DECREASED	4.6	DECREASED	NO	YES	58.2	NORMAL	54.8
6	DS	663471	NO RESPONSE	2.7	NORMAL	YES	NO	YES	YES	NO	4.9	INCREASED	4.5	INCREASED	NO	YES	6.7	NORMAL	10.2	NORMAL	NO	NO	38.7	DECREASED	39.7
7	R	667314	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES	7	INCREASED	9	INCREASED	NO	YES	1.5	DECREASED	1	DECREASED	NO	YES	47.9	NORMAL	48.4
8	HS	678065	NORMAL	3.5	NORMAL	NO	NO	YES	YES	NO	3.9	NORMAL	4.5	INCREASED	YES	NO	2.3	DECREASED	1.5	DECREASED	NO	YES	54.8	NORMAL	50
9	PG	677371	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES	4.8	INCREASED	6.2	INCREASED	NO	YES	7.1	NORMAL	6	NORMAL	NO	NO	50.5	NORMAL	46
10	NK	679377	NORMAL	4.4	NORMAL	NO	NO	YES	YES	NO	15.5	INCREASED	23	INCREASED	NO	YES	1.7	DECREASED	1.2	DECREASED	NO	YES	35.1	DECREASED	34.6
11	MK	698042	NORMAL	6.5	NORMAL	NO	NO	NO	NO	NO	2.6	NORMAL	2.4	NORMAL	NO	NO	12	NORMAL	9.1	NORMAL	NO	NO	62.2	NORMAL	61.3
12	DK	702439	NORMAL	1.8	NORMAL	NO	NO	YES	YES	NO	3.3	NORMAL	3.9	NORMAL	NO	NO	11	NORMAL	6.3	NORMAL	NO	NO	64.8	NORMAL	63.9
13	NG	712126	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES	3.2	NORMAL	4.1	NORMAL	NO	NO	4.4	DECREASED	4	DECREASED	NO	YES	58.2	NORMAL	58.2
14	ES	715561	NORMAL	7.7	NORMAL	NO	NO	YES	YES	NO	3.1	NORMAL	4	NORMAL	NO	NO	11.2	NORMAL	5.9	NORMAL	NO	NO	63	NORMAL	58.2
15	TL	722336	NORMAL	8.4	INCREASED	YES	NO	NO	YES	NO	3.1	NORMAL	3.1	NORMAL	NO	NO	9.2	NORMAL	8.7	NORMAL	NO	NO	56.1	NORMAL	54.1
16	HK	726934	NORMAL	4.3	NORMAL	NO	NO	NO	NO	NO	2.3	NORMAL	2.4	NORMAL	NO	NO	12.1	NORMAL	10.8	NORMAL	NO	NO	68.7	NORMAL	66.7
17	RB	726888	NORMAL	5.4	NORMAL	NO	NO	YES	YES	NO	3.2	NORMAL	3	NORMAL	NO	NO	10.2	NORMAL	12.1	NORMAL	NO	NO	56.8	NORMAL	60.5
18	JK	727668	NORMAL	5.6	NORMAL	NO	NO	YES	YES	NO	3.2	NORMAL	3.8	NORMAL	NO	NO	1.1	DECREASED	1.3	DECREASED	NO	YES		NO RESPONSE	
19	SS	728987	NO RESPONSE	15.3	INCREASED	NO	YES	YES	YES	YES	3.8	NORMAL	3.4	NORMAL	NO	NO	5.4	NORMAL	4.2	DECREASED	YES	NO	50.5	NORMAL	52
20	S	734436	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
21	SS	736396	NO RESPONSE	6.8	NORMAL	YES	NO	YES	YES	NO		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
22	GS	741216	NORMAL	5.5	NORMAL	NO	NO	YES	YES	NO	3.2	NORMAL	3.3	NORMAL	NO	NO	3.4	DECREASED	4.5	DECREASED	NO	YES	59.7	NORMAL	52.3
23	HS	765811	NORMAL	5.9	NORMAL	NO	NO	YES	YES	NO	4.8	INCREASED	5.9	INCREASED	NO	YES	1.9	DECREASED	2.2	DECREASED	NO	YES	49	NORMAL	50.5
24	BS	769025	INCREASED	13	INCREASED	NO	YES	YES	YES	YES		NO RESPONSE	11.9	INCREASED	NO	YES		NO RESPONSE	0.6	DECREASED	NO	YES		NO RESPONSE	28.8
25	PCG	752729	INCREASED	5.6	NORMAL	YES	NO	YES	YES	NO	4.3	NORMAL	4.9	INCREASED	YES	NO	5	NORMAL	3.6	DECREASED	YES	NO	42.6	DECREASED	33.6
26	VR	783712	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
27	SK	803750	NORMAL	1.9	NORMAL	NO	NO	YES	YES	NO	4.3	NORMAL	3.1	NORMAL	NO	NO	2	DECREASED	2.7	DECREASED	NO	YES	57.5	NORMAL	56.1
28	AK	823722	INCREASED	7.2	NORMAL	YES	NO	YES	YES	NO	5.1	INCREASED	4.9	INCREASED	NO	YES	2.1	DECREASED	1.6	DECREASED	NO	YES	39.3	DECREASED	38.7
29	RK	979343	NORMAL	3.6	NORMAL	NO	NO	YES	YES	NO	3.5	NORMAL	3.2	NORMAL	NO	NO	8.2	NORMAL	6.3	NORMAL	NO	NO	61.3	NORMAL	54.8
30	RK	843997	NORMAL	3.4	NORMAL	NO	NO	YES	YES	NO	4.1	NORMAL	3.9	NORMAL	NO	NO	9.1	NORMAL	5.5	NORMAL	NO	NO	39.3	DECREASED	37.1
31	LD	848012	INCREASED	9.9	INCREASED	NO	YES	YES	YES	YES	7.5	INCREASED	7.7	INCREASED	NO	YES	2.6	DECREASED	1.5	DECREASED	NO	YES	36.8	DECREASED	31.3
32	MK	868747	NORMAL	4.8	NORMAL	NO	NO	NO	NO	NO	5	INCREASED	4	NORMAL	YES	NO	6.6	NORMAL	8.2	NORMAL	NO	NO	63.9	NORMAL	59.7
33	BDS	869851	INCREASED	8.6	INCREASED	NO	YES	YES	YES	YES	4.8	INCREASED	5.7	INCREASED	NO	YES	4.1	DECREASED	5.8	NORMAL	YES	NO	37.7	DECREASED	41.1

S.NO	NAME	MRD NO.	UPPER LIMB																							
			MEDIAN NERVE									SENSORY												ONSET LAT		
			LOCITY (>45 m/s)			F- WAVE LATENCY (<31.2 ms)				F- WAVE PERSISTENCE (>50%)		PEAK LATENCY (<3.6 ms)				AMPLITUDE (>11 microV)				VELOCITY (≥50 m/s)				WRIST		
			RIGHT	U/L INVOLVEME	B/L INVOLVEME	LEFT		RIGHT		LEFT	RIGHT	LEFT		RIGHT		LEFT		RIGHT		LEFT		RIGHT		LEFT		RI
1	AK	620103	NORMAL	NO	NO	23.8	NORMAL	25.7	NORMAL	NORMAL	NORMAL	2.7	NORMAL	2.9	NORMAL	24.1	NORMAL	28.6	NORMAL	66	NORMAL	66	NORMAL	2.4	NORMAL	2.5
2	BK	623416	NO RESPONSE	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	4	INCREASED	3.8
3	SS	638200	NORMAL	NO	NO	29.6	NORMAL	28	NORMAL	DECREASED	NORMAL	4.2	INCREASED	3.5	NORMAL	35.3	NORMAL	34.8	NORMAL	42.2	DECREASED	50.4	NORMAL	3.5	INCREASED	2.3
4	SK	646844	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	7.1	INCREASED	7.6
5	AK	660069	NORMAL	NO	NO	36.6	INCREASED	33.9	INCREASED	NORMAL	NORMAL	4.1	INCREASED	3.7	INCREASED	27.2	NORMAL	26.7	NORMAL	45.2	DECREASED	51.5	NORMAL	2.8	NORMAL	3.3
6	DS	663471	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	28.9	NORMAL	DECREASED	DECREASED	3.3	NORMAL	3.2	NORMAL	61.4	NORMAL	50.3	NORMAL	56.5	NORMAL	55.6	NORMAL	3.3	NORMAL	4.7
7	R	667314	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	4.8	INCREASED	5.5
8	HS	678065	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	3.1	NORMAL	50.8	NORMAL	37.5	NORMAL	68	NORMAL	57.4	NORMAL		NO RESPONSE	
9	PG	677371	NORMAL	NO	NO	29.3	NORMAL	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.5	NORMAL	3.8	INCREASED	78.5	NORMAL	58.5	NORMAL	51.1	NORMAL	52.2	NORMAL	3.6	INCREASED	5.2
10	NK	679377	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	4.7	INCREASED	3.6
11	MK	698042	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.6	NORMAL	3	NORMAL	61.4	NORMAL	39.8	NORMAL	68	NORMAL	64.8	NORMAL	2.4	NORMAL	2.5
12	DK	702439	NORMAL	NO	NO	28.3	NORMAL	28.8	NORMAL	DECREASED	DECREASED	2.5	NORMAL	2.5	NORMAL	75.9	NORMAL	60.8	NORMAL	70.7	NORMAL	71.4	NORMAL	2.9	NORMAL	2.8
13	NG	712126	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.8	NORMAL	3	NORMAL	60.2	NORMAL	43.7	NORMAL	70.7	NORMAL	64.2	NORMAL	2.5	NORMAL	2.7
14	ES	715561	NORMAL	NO	NO	27.2	NORMAL	24.2	NORMAL	DECREASED	NORMAL	2.9	NORMAL	2.7	NORMAL	96.6	NORMAL	84.6	NORMAL	69.3	NORMAL	72.9	NORMAL	3.4	NORMAL	2.6
15	TL	722336	NORMAL	NO	NO	33.1	INCREASED	30.6	NORMAL	DECREASED	DECREASED	2.9	NORMAL	3.1	NORMAL	28	NORMAL	31.5	NORMAL	65.4	NORMAL	66.7	NORMAL	2.7	NORMAL	2.6
16	HK	726934	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.4	NORMAL	2.6	NORMAL	79.1	NORMAL	51.3	NORMAL	75.3	NORMAL	78.7	NORMAL	1.5	NORMAL	1.8
17	RB	726888	NORMAL	NO	NO	25.2	NORMAL	25.3	NORMAL	NORMAL	NORMAL	2.9	NORMAL	2.7	NORMAL	96.7	NORMAL	100.4	NORMAL	63.1	NORMAL	67.3	NORMAL	2.8	NORMAL	3.8
18	JK	727668	NO RESPONSE	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.8	NORMAL	3	NORMAL	36	NORMAL	35.1	NORMAL	70.7	NORMAL	65.4	NORMAL	2.4	NORMAL	2.6
19	SS	728987	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	2.9	NORMAL	40.1	NORMAL	30.7	NORMAL	62.5	NORMAL	60.3	NORMAL	2.6	NORMAL	2.6
20	S	734436	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.3	NORMAL	3	NORMAL	43.1	NORMAL	39.8	NORMAL	56.5	NORMAL	64.2	NORMAL		NO RESPONSE	
21	SS	736396	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.2	NORMAL	3.3	NORMAL	25.9	NORMAL	22.8	NORMAL	56.5	NORMAL	56	NORMAL		NO RESPONSE	
22	GS	741216	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	2.6	NORMAL	33.7	NORMAL	35.3	NORMAL	66	NORMAL	70	NORMAL	2.4	NORMAL	2.7
23	HS	765811	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.3	NORMAL	3.5	NORMAL	51.8	NORMAL	41.1	NORMAL	61.4	NORMAL	50.7	NORMAL	3.3	NORMAL	3.9
24	BS	769025	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	
25	PCG	752729	DECREASED	NO	YES	38.7	INCREASED	44	INCREASED	DECREASED	NORMAL	4.1	INCREASED	4.3	INCREASED	28	NORMAL	25.1	NORMAL	43.8	DECREASED	44.3	DECREASED	4.1	INCREASED	3.4
26	VR	783712	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.9	NORMAL	3.2	NORMAL	13	NORMAL	12.1	NORMAL	65.4	NORMAL	61.4	NORMAL		NO RESPONSE	
27	SK	803750	NORMAL	NO	NO	23.8	NORMAL	27.9	NORMAL	NORMAL	NORMAL	2.8	NORMAL	2.9	NORMAL	59	NORMAL	67.2	NORMAL	64.8	NORMAL	61.4	NORMAL	2.6	NORMAL	2.6
28	AK	823722	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	2.9	NORMAL	3.6
29	RK	979343	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.3	NORMAL	3	NORMAL	20.7	NORMAL	19.9	NORMAL	54.3	NORMAL	64.2	NORMAL	2.4	NORMAL	2.2
30	RK	843997	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.3	INCREASED	4.8	INCREASED	15.1	NORMAL	17.4	NORMAL	53.4	NORMAL	41.9	DECREASED	2.6	NORMAL	2.8
31	LD	848012	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	8	INCREASED	5.5
32	MK	868747	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.7	INCREASED	4	INCREASED	23.1	NORMAL	14.9	NORMAL	37.4	DECREASED	42.9	DECREASED	2.9	NORMAL	2.2
33	BDS	869851	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.3	INCREASED	5.3	INCREASED	13.9	NORMAL	18.1	NORMAL	38.5	DECREASED	34.7	DECREASED	2.7	NORMAL	3.2

S.NO	NAME	MRD NO.	ULNAR NERVE																							
			MOTOR																F- WAVE LATENCY (<32.1 ms)				F- WAVE PERSISTENCE (>50%)		PEAK LATENCY (<3.0 ms)		
			ENCY (<3.5 ms)			AMPLITUDE (>5.5 mV)						VELOCITY (>43 m/s)															
						WRIST																					
			GHT	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT		RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT		RIGHT		U/L INVOLVEME	B/L INVOLVEME	LEFT		RIGHT		LEFT	RIGHT	LEFT		RI
1	AK	620103	NORMAL	NO	NO	3.9	DECREASED	2.5	DECREASED	NO	YES	67.6	NORMAL	66.7	NORMAL	NO	NO	24.1	NORMAL	24.6	NORMAL	DECREASED	NORMAL	2.8	NORMAL	2.7
2	BK	623416	INCREASED	NO	YES	2.2	DECREASED	2.4	DECREASED	NO	YES	43.1	NORMAL	59.3	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	27	NORMAL	DECREASED	NORMAL		NO RESPONSE	
3	SS	638200	NORMAL	YES	NO	8.4	NORMAL	9.8	NORMAL	NO	NO	57.8	NORMAL	48	NORMAL	NO	NO	31.4	NORMAL	29.1	NORMAL	DECREASED	DECREASED	3.2	INCREASED	3.2
4	SK	646844	INCREASED	NO	YES	2.3	DECREASED	2.3	DECREASED	NO	YES	59.3	NORMAL	55.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE	
5	AK	660069	NORMAL	NO	NO	7.1	NORMAL	8.7	NORMAL	NO	NO	54.5	NORMAL	49	NORMAL	NO	NO	29.7	NORMAL	26.1	NORMAL	NORMAL	NORMAL	3.9	INCREASED	3.7
6	DS	663471	INCREASED	YES	NO	4.3	DECREASED	6	NORMAL	YES	NO	38.4	DECREASED	37.2	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	38.5	INCREASED	DECREASED	DECREASED	3.4	INCREASED	3.2
7	R	667314	INCREASED	NO	YES	2.6	DECREASED	1.5	DECREASED	NO	YES	67.6	NORMAL	60.8	NORMAL	NO	NO	36	INCREASED	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.3	INCREASED	
8	HS	678065	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.3	NORMAL	2.5
9	PG	677371	INCREASED	NO	YES	6.3	NORMAL	4	DECREASED	YES	NO	69.3	NORMAL	43.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.6	INCREASED	3.8
10	NK	679377	INCREASED	NO	YES	2.8	DECREASED	4	DECREASED	NO	YES	61.5	NORMAL	50.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.8	INCREASED	3.5
11	MK	698042	NORMAL	NO	NO	12.6	NORMAL	10.3	NORMAL	NO	NO	80	NORMAL	76.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.4	NORMAL	2.7
12	DK	702439	NORMAL	NO	NO	2.3	DECREASED	1.8	DECREASED	NO	YES	64.9	NORMAL	58.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.5	NORMAL	2.2
13	NG	712126	NORMAL	NO	NO	2.2	DECREASED	3.4	DECREASED	NO	YES	57.8	NORMAL	54.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.3	NORMAL	2.5
14	ES	715561	NORMAL	NO	NO	8.9	NORMAL	7.2	NORMAL	NO	NO	67.6	NORMAL	63.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.6	NORMAL	2.7
15	TL	722336	NORMAL	NO	NO	5.5	NORMAL	3.6	DECREASED	YES	NO	71.6	NORMAL	63.2	NORMAL	NO	NO	31	NORMAL	33	INCREASED	DECREASED	DECREASED	3	NORMAL	3
16	HK	726934	NORMAL	NO	NO	8.7	NORMAL	7.2	NORMAL	NO	NO	71.6	NORMAL	77.4	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.3	NORMAL	2.2
17	RB	726888	INCREASED	YES	NO	6.3	NORMAL	5	DECREASED	YES	NO	72.7	NORMAL	82.1	NORMAL	NO	NO	28.7	NORMAL	28.5	NORMAL	DECREASED	NORMAL	2.2	NORMAL	2.4
18	JK	727668	NORMAL	NO	NO	2.3	DECREASED	2.5	DECREASED	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.4	NORMAL	3
19	SS	728987	NORMAL	NO	NO	5.3	DECREASED	4	DECREASED	NO	YES	55.6	NORMAL	46.8	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.8	NORMAL	2.7
20	S	734436	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.8	NORMAL	2.6
21	SS	736396	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.6	INCREASED	2.9
22	GS	741216	NORMAL	NO	NO	2.6	DECREASED	3.3	DECREASED	NO	YES	61.5	NORMAL	69.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	2.3
23	HS	765811	INCREASED	YES	NO	1.4	DECREASED	2.6	DECREASED	NO	YES	50.5	NORMAL	50	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	3.2
24	BS	769025	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE	
25	PCG	752729	NORMAL	YES	NO	3.1	DECREASED	4.2	DECREASED	NO	YES	44.9	NORMAL	45.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.7	INCREASED	3.7
26	VR	783712	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.2	NORMAL	2.3
27	SK	803750	NORMAL	NO	NO	9.6	NORMAL	6.2	NORMAL	NO	NO	63.2	NORMAL	69.6	NORMAL	NO	NO	31.3	NORMAL	28.4	NORMAL	DECREASED	DECREASED	2.8	NORMAL	2.8
28	AK	823722	INCREASED	YES	NO	3.6	DECREASED	4.1	DECREASED	NO	YES	51.2	NORMAL	57.1	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	
29	RK	979343	NORMAL	NO	NO	4.1	DECREASED	5.5	NORMAL	YES	NO	63.2	NORMAL	54.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.1	INCREASED	2.6
30	RK	843997	NORMAL	NO	NO	3.9	DECREASED	4.4	DECREASED	NO	YES	48	NORMAL	44	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	INCREASED	
31	LD	848012	INCREASED	NO	YES	2.6	DECREASED	1.4	DECREASED	NO	YES	43.6	NORMAL	32.9	DECREASED	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE	
32	MK	868747	NORMAL	NO	NO	8.2	NORMAL	7.4	NORMAL	NO	NO	60.8	NORMAL	44.1	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.4	INCREASED	2.6
33	BDS	869851	NORMAL	NO	NO	4.5	DECREASED	5.1	DECREASED	NO	YES	48	NORMAL	46.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	INCREASED	3.2

S.NO	NAME	MRD NO.	SENSORY									MOTOR															PERONEAL NERVE		
				AMPLITUDE (>11 microV)				VELOCITY (≥50 m/s)				ONSET LATENCY (<5.4 ms)						AMPLITUDE (>2.0 mV)							VEL				
												ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT					
			GHT	LEFT		RIGHT		LEFT		RIGHT		LEFT		RIGHT				LEFT		RIGHT				LEFT		F			
1	AK	620103	NORMAL	27.8	NORMAL	25.2	NORMAL	64.8	NORMAL	75.3	NORMAL	4.5	NORMAL	3.7	NORMAL	NO	NO	1.8	DECREASED	2	DECREASED	NO	YES	52.6	NORMAL	43.8			
2	BK	623416	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	11.5	INCREASED	12.2	INCREASED	NO	YES	0.4	DECREASED	0.4	DECREASED	NO	YES	40.3	NORMAL	28.7			
3	SS	638200	INCREASED	31.3	NORMAL	27.1	NORMAL	59.3	NORMAL	59.8	NORMAL	6.1	INCREASED	6.9	INCREASED	NO	YES	6.8	NORMAL	6.4	NORMAL	NO	NO	34.2	DECREASED	34			
4	SK	646844	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
5	AK	660069	INCREASED	20.4	NORMAL	20.8	NORMAL	48.3	DECREASED	51.5	NORMAL	9.2	INCREASED	9	INCREASED	NO	YES	5.3	NORMAL	6.1	NORMAL	NO	NO	47.2	NORMAL	39.2			
6	DS	663471	INCREASED	41.1	NORMAL	32.2	NORMAL	51.1	NORMAL	59.8	NORMAL	7	INCREASED	10	INCREASED	NO	YES	2.7	NORMAL	3.3	NORMAL	NO	NO	29	DECREASED	27.3			
7	R	667314	NO RESPONSE	6	DECREASED		NO RESPONSE	59.8	NORMAL		NO RESPONSE	16	INCREASED	12.9	INCREASED	NO	YES	0.7	DECREASED	0.9	DECREASED	NO	YES	53.1	NORMAL	45.5			
8	HS	678065	NORMAL	46.7	NORMAL	37.3	NORMAL	79.5	NORMAL	72.2	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
9	PG	677371	INCREASED	78.5	NORMAL	58.5	NORMAL	51.1	NORMAL	52.2	NORMAL	9.3	INCREASED	8	INCREASED	NO	YES	4.5	NORMAL	6.8	NORMAL	NO	NO	34.5	DECREASED	31.9			
10	NK	679377	INCREASED	16	NORMAL	20.7	NORMAL	50.4	NORMAL	56.9	NORMAL	6.7	INCREASED	10.7	INCREASED	NO	YES	3.6	NORMAL	1.9	DECREASED	YES	NO	41.7	NORMAL	33.1			
11	MK	698042	NORMAL	37	NORMAL	33.5	NORMAL	76.1	NORMAL	63.6	NORMAL	3.6	NORMAL	4.2	NORMAL	NO	NO	4.9	NORMAL	7.7	NORMAL	NO	NO	52.6	NORMAL	53.8			
12	DK	702439	NORMAL	67	NORMAL	56.9	NORMAL	71.4	NORMAL	83.3	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
13	NG	712126	NORMAL	53.9	NORMAL	45.4	NORMAL	78.7	NORMAL	80.5	NORMAL	4.5	NORMAL	4.3	NORMAL	NO	NO	1	DECREASED	1.5	DECREASED	NO	YES	45.8	NORMAL	45.8			
14	ES	715561	NORMAL	96.6	NORMAL	84.6	NORMAL	69.3	NORMAL	72.9	NORMAL	4	NORMAL	5.2	NORMAL	NO	NO	3.8	NORMAL	3	NORMAL	NO	NO	47.2	NORMAL	43.8			
15	TL	722336	NORMAL	19.7	NORMAL	22.6	NORMAL	68	NORMAL	61.4	NORMAL	4.8	NORMAL	5.4	NORMAL	NO	NO	2.8	NORMAL	2.6	NORMAL	NO	NO	35.3	DECREASED	43.2			
16	HK	726934	NORMAL	78.7	NORMAL	78.1	NORMAL	81.4	NORMAL	82.4	NORMAL	3.1	NORMAL	3.8	NORMAL	NO	NO	7.3	NORMAL	4.9	NORMAL	NO	NO	51.3	NORMAL	45			
17	RB	726888	NORMAL	97.1	NORMAL	87.6	NORMAL	86.4	NORMAL	83.3	NORMAL	5.4	NORMAL	5.2	NORMAL	NO	NO	1.6	DECREASED	0.5	DECREASED	NO	YES	54.1	NORMAL	49.6			
18	JK	727668	NORMAL	36	NORMAL	35.1	NORMAL	70.7	NORMAL	65.4	NORMAL	4.4	NORMAL	4.3	NORMAL	NO	NO	1.6	DECREASED	1.4	DECREASED	NO	YES	42.9	NORMAL	40.5			
19	SS	728987	NORMAL	19.4	NORMAL	20.6	NORMAL	67.3	NORMAL	66.7	NORMAL	6.3	INCREASED	5.9	INCREASED	NO	YES	3.6	NORMAL	4.3	NORMAL	NO	NO	36.4	DECREASED	32.3			
20	S	734436	NORMAL	36.6	NORMAL	44.3	NORMAL	70	NORMAL	85.4	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
21	SS	736396	NORMAL	21.4	NORMAL	20.5	NORMAL	53.4	NORMAL	75.3	NORMAL	5.3	NORMAL	4.9	NORMAL	NO	NO	0.6	DECREASED	0.5	DECREASED	NO	YES	40.5	NORMAL	43.2			
22	GS	741216	NORMAL	28.9	NORMAL	23.1	NORMAL	67.3	NORMAL	77.8	NORMAL	5.2	NORMAL	5.1	NORMAL	NO	NO	1.2	DECREASED	0.5	DECREASED	NO	YES	44.8	NORMAL	47.2			
23	HS	765811	INCREASED	37.5	NORMAL	48.8	NORMAL	64.2	NORMAL	61.9	NORMAL	5.4	NORMAL	5.5	INCREASED	YES	NO	0.4	DECREASED	0.7	DECREASED	NO	YES	24.3	DECREASED	22.7			
24	BS	769025	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
25	PCG	752729	INCREASED	26.2	NORMAL	16.8	NORMAL	47.9	DECREASED	52.2	NORMAL	5.9	INCREASED	4.8	NORMAL	YES	NO	2.7	NORMAL	2.3	NORMAL	NO	NO	29.9	DECREASED	31.1			
26	VR	783712	NORMAL	20.2	NORMAL	19.6	NORMAL	88.6	NORMAL	84.3	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE				
27	SK	803750	NORMAL	41.8	NORMAL	43.3	NORMAL	66.7	NORMAL	66	NORMAL	4.5	NORMAL	5	NORMAL	NO	NO	6.4	NORMAL	5.4	NORMAL	NO	NO	44.8	NORMAL	46.9			
28	AK	823722	NO RESPONSE	13.4	NORMAL		NO RESPONSE	68.6	NORMAL		NO RESPONSE	5.8	INCREASED	5.2	NORMAL	YES	NO	1	DECREASED	1.5	DECREASED	NO	YES	45.1	NORMAL	40			
29	RK	979343	NORMAL	11.9	NORMAL	13.2	NORMAL	54.3	NORMAL	72.9	NORMAL	3.6	NORMAL	3.9	NORMAL	NO	NO	4.2	NORMAL	3.3	NORMAL	NO	NO	43	NORMAL	38.3			
30	RK	843997	NO RESPONSE	12.4	NORMAL		NO RESPONSE	59.3	NORMAL		NO RESPONSE	6.3	INCREASED	9.7	INCREASED	NO	YES	3.4	NORMAL	2.3	NORMAL	NO	NO	36.8	DECREASED	38.8			
31	LD	848012	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	9	INCREASED	NO	YES		NO RESPONSE	0.6	DECREASED	NO	YES		NO RESPONSE	30.6			
32	MK	868747	NORMAL	33.8	NORMAL	27.5	NORMAL	56.9	NORMAL	68.6	NORMAL	3.5	NORMAL	4	NORMAL	NO	NO	4	NORMAL	3.9	NORMAL	NO	NO	44.1	NORMAL	43.8			
33	BDS	869851	INCREASED	17.6	NORMAL	19.1	NORMAL	59.3	NORMAL	63.6	NORMAL	5.7	INCREASED	6.4	INCREASED	NO	YES	4.5	NORMAL	3.1	NORMAL	NO	NO	28	DECREASED	26.4			

S.NO	NAME	MRD NO.	LOWER LIMB																					
												TIBIAL NER												
												MOTOR												
			OCITY (≥37 m/s)			F- WAVE LATENCY (<59.3 ms)				F- WAVE PERSISTENCE (>30%)		ONSET LATENCY (<6.1 ms)						AMPLITUDE (>3.5 mV)						
												ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT
			IGHT	U/L INVOLVEMENT	B/L INVOLVEMENT	LEFT		RIGHT		LEFT	RIGHT	LEFT		RIGHT				LEFT		RIGHT			
1	AK	620103	NORMAL	NO	NO	44.8	NORMAL	45	NORMAL	NORMAL	NORMAL	4	NORMAL	3.7	NORMAL	NO	NO	2.6	DECREASED	3	DECREASED	NO	YES
2	BK	623416	DECREASED	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE	9.6	INCREASED	NO	YES		NO RESPONSE	0.4	DECREASED	NO	YES
3	SS	638200	DECREASED	NO	YES	61.2	INCREASED	62.7	INCREASED	NORMAL	NORMAL	6.5	INCREASED	7.2	INCREASED	NO	YES	8.2	NORMAL	9.3	NORMAL	NO	NO
4	SK	646844	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
5	AK	660069	NORMAL	NO	NO	54	NORMAL	64.4	INCREASED	DECREASED	NORMAL	7.4	INCREASED	7.3	INCREASED	NO	YES	13.2	NORMAL	9.5	NORMAL	NO	NO
6	DS	663471	DECREASED	NO	YES	67.1	INCREASED	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	10	INCREASED	13.4	INCREASED	NO	YES	2.8	DECREASED	0.9	DECREASED	NO	YES
7	R	667314	NORMAL	NO	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	10.9	INCREASED	10	INCREASED	NO	YES	2.2	DECREASED	1.5	DECREASED	NO	YES
8	HS	678065	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
9	PG	677371	DECREASED	NO	YES	58.2	NORMAL	55.8	NORMAL	NORMAL	NORMAL	7.8	INCREASED	8.7	INCREASED	NO	YES	5.3	NORMAL	4.6	NORMAL	NO	NO
10	NK	679377	DECREASED	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	7.5	INCREASED	7.6	INCREASED	NO	YES	2.9	DECREASED	2.2	DECREASED	NO	YES
11	MK	698042	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.3	NORMAL	3.6	NORMAL	NO	NO	18.3	NORMAL	16.6	NORMAL	NO	NO
12	DK	702439	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	5.6	NORMAL	7.8	INCREASED	YES	NO	1.1	DECREASED	0.9	DECREASED	NO	YES
13	NG	712126	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.3	NORMAL	4.8	NORMAL	NO	NO	0.8	DECREASED	0.6	DECREASED	NO	YES
14	ES	715561	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.2	NORMAL	4	NORMAL	NO	NO	6.4	NORMAL	6.8	NORMAL	NO	NO
15	TL	722336	NORMAL	YES	NO	52.1	NORMAL	51.4	NORMAL	NORMAL	NORMAL	4.1	NORMAL	3.8	NORMAL	NO	NO	6.3	NORMAL	9.6	NORMAL	NO	NO
16	HK	726934	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	NORMAL	3.7	NORMAL	NO	NO	12.3	NORMAL	16.2	NORMAL	NO	NO
17	RB	726888	NORMAL	NO	NO	49.8	NORMAL	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	6	NORMAL	5.8	NORMAL	NO	NO	1.3	DECREASED	1	DECREASED	NO	YES
18	JK	727668	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	5.3	NORMAL	5.6	NORMAL	NO	NO	4.1	NORMAL	3	DECREASED	YES	NO
19	SS	728987	DECREASED	NO	YES	60.4	INCREASED	54.7	NORMAL	DECREASED	NORMAL	4.7	NORMAL	4.5	NORMAL	NO	NO	3.9	NORMAL	2.1	DECREASED	YES	NO
20	S	734436	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
21	SS	736396	NORMAL	NO	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	5.5	NORMAL	5.7	NORMAL	NO	NO	0.4	DECREASED	0.7	DECREASED	NO	YES
22	GS	741216	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	5.3	NORMAL	5.4	NORMAL	NO	NO	2.4	DECREASED	1.5	DECREASED	NO	YES
23	HS	765811	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
24	BS	769025	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
25	PCG	752729	DECREASED	NO	YES	71.7	INCREASED	70.3	INCREASED	NORMAL	NORMAL	6.1	NORMAL	6.8	INCREASED	YES	NO	5.2	NORMAL	8	NORMAL	NO	NO
26	VR	783712	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
27	SK	803750	NORMAL	NO	NO	47.2	NORMAL	49.2	NORMAL	NORMAL	NORMAL	6	NORMAL	5.9	NORMAL	NO	NO	8.8	NORMAL	8.8	NORMAL	NO	NO
28	AK	823722	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE	5.3	NORMAL	YES	NO		NO RESPONSE	4	NORMAL	YES	NO
29	RK	979343	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.6	NORMAL	3.6	NORMAL	NO	NO	9.9	NORMAL	8.4	NORMAL	NO	NO
30	RK	843997	NORMAL	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	5.3	NORMAL	4.6	NORMAL	NO	NO	1.9	DECREASED	3	DECREASED	NO	YES
31	LD	848012	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
32	MK	868747	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.1	NORMAL	4.5	NORMAL	NO	NO	5.4	NORMAL	5.2	NORMAL	NO	NO
33	BDS	869851	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	7.1	INCREASED	6.8	INCREASED	NO	YES	3.5	NORMAL	3.7	NORMAL	NO	NO

S.NO	NAME	MRD NO.	VE												SURAL NERVE									
			VELOCITY (>35 m/s)						F- WAVE LATENCY (<54 ms)				F- WAVE PERSISTENCE (>50%)		SENSORY									
															PEAK LATENCY (<3.1 ms)				AMPLITUDE (>9.0 microV)				VELOCITY	
			LEFT		RIGHT		U/L INVOLVEMENT	B/L INVOLVEMENT	LEFT		RIGHT		LEFT	RIGHT	LEFT		RIGHT		LEFT		RIGHT		LEFT	
1	AK	620103	45.7	NORMAL	40.8	NORMAL	NO	NO	44.2	NORMAL	44.7	NORMAL	DECREASED	DECREASED	3.3	INCREASED	3.1	INCREASED	18.5	NORMAL	17.5	NORMAL	62.5	NORMAL
2	BK	623416		NO RESPONSE	33	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.2	INCREASED	3.1	INCREASED	8.8	DECREASED	10.3	NORMAL	58.3	NORMAL
3	SS	638200	28.9	DECREASED	32.4	DECREASED	NO	YES	60.5	INCREASED	69.8	INCREASED	NORMAL	DECREASED	4.9	INCREASED	4.3	INCREASED	21.2	NORMAL	22.1	NORMAL	44.9	NORMAL
4	SK	646844		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE
5	AK	660069	32.3	DECREASED	35.4	NORMAL	YES	NO	49.6	NORMAL	58.5	INCREASED	DECREASED	DECREASED	3.9	INCREASED	4.1	INCREASED	30.9	NORMAL	29.8	NORMAL	43.5	NORMAL
6	DS	663471	25.8	DECREASED	26.7	DECREASED	NO	YES	68.2	INCREASED	NOT RECORDED	NOT RECORDED	NORMAL	NOT RECORDED	4.5	INCREASED	4.3	INCREASED	12.6	NORMAL	16.1	NORMAL	41.4	NORMAL
7	R	667314	32.8	DECREASED	36	NORMAL	YES	NO	61.7	INCREASED	59.9	INCREASED	NORMAL	DECREASED	3.7	INCREASED	3.6	INCREASED	28.9	NORMAL	29	NORMAL	52.6	NORMAL
8	HS	678065		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.3	INCREASED	3.4	INCREASED	28.4	NORMAL	23.8	NORMAL	50.7	NORMAL
9	PG	677371	34	DECREASED	27.4	DECREASED	NO	YES	57.7	INCREASED	61	INCREASED	NORMAL	DECREASED	4.1	INCREASED	4.1	INCREASED	21	NORMAL	25.7	NORMAL	41.9	NORMAL
10	NK	679377	34.2	DECREASED	31.7	DECREASED	NO	YES	60.4	INCREASED	NO RESPONSE	NO RESPONSE	NORMAL	DECREASED	3.7	INCREASED	3.6	INCREASED	30.7	NORMAL	24.3	NORMAL	54.7	NORMAL
11	MK	698042	42.6	NORMAL	49.6	NORMAL	NO	NO	48.1	NORMAL	50	NORMAL	DECREASED	DECREASED	2.9	NORMAL	3	NORMAL	35.4	NORMAL	37.8	NORMAL	63.1	NORMAL
12	DK	702439	29	DECREASED	32	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	2.6	NORMAL	2.4	NORMAL	49.4	NORMAL	40.1	NORMAL	74.5	NORMAL
13	NG	712126	32.2	DECREASED	40.8	NORMAL	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.6	NORMAL	2.6	NORMAL	23.8	NORMAL	23.9	NORMAL	68	NORMAL
14	ES	715561	40	NORMAL	33.3	DECREASED	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.4	NORMAL	2.3	NORMAL	30.1	NORMAL	30.3	NORMAL	75.3	NORMAL
15	TL	722336	34.4	DECREASED	36.4	NORMAL	YES	NO	55	INCREASED	53.7	NORMAL	DECREASED	DECREASED	3.1	NORMAL	3.5	INCREASED	16.1	NORMAL	13.7	NORMAL	56.9	NORMAL
16	HK	726934		NO RESPONSE	45.1	NORMAL	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.5	NORMAL	2.6	NORMAL	37	NORMAL	27.2	NORMAL	77.8	NORMAL
17	RB	726888	47.8	NORMAL	38.8	NORMAL	NO	NO	49.9	NORMAL	NOT RECORDED	NOT RECORDED	NORMAL	NOT RECORDED	2.8	NORMAL	2.7	NORMAL	21.8	NORMAL	17.1	NORMAL	66.7	NORMAL
18	JK	727668	33.2	DECREASED	35.7	NORMAL	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	INCREASED	3.5	INCREASED	29.9	NORMAL	25.9	NORMAL	54.3	NORMAL
19	SS	728987	31.2	DECREASED	37.6	NORMAL	YES	NO	62.6	INCREASED	57.6	INCREASED	DECREASED	DECREASED	3.8	INCREASED	3.6	INCREASED	13.5	NORMAL	14.4	NORMAL	49.3	NORMAL
20	S	734436		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.1	NORMAL	3.1	NORMAL	44.6	NORMAL	41.1	NORMAL	58.3	NORMAL
21	SS	736396	41	NORMAL	30.3	DECREASED	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.9	NORMAL	2.8	NORMAL	34.9	NORMAL	34.2	NORMAL	71.4	NORMAL
22	GS	741216	37.2	NORMAL	40.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.9	NORMAL	2.9	NORMAL	17.3	NORMAL	13.7	NORMAL	63.1	NORMAL
23	HS	765811		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.6	INCREASED	3.6	INCREASED	40.4	NORMAL	38.4	NORMAL	53	NORMAL
24	BS	769025		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE
25	PCG	752729	27.1	DECREASED	31.2	DECREASED	NO	YES	78.3	INCREASED	70.5	INCREASED	NORMAL	NORMAL	4.2	INCREASED	3.6	INCREASED	11.4	NORMAL	9.4	NORMAL	47	NORMAL
26	VR	783712		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.7	NORMAL	2.4	NORMAL	21.2	NORMAL	19.1	NORMAL	65.4	NORMAL
27	SK	803750	47.8	NORMAL	43.5	NORMAL	NO	NO	49.9	NORMAL	50	NORMAL	DECREASED	NORMAL	3.1	NORMAL	3.4	INCREASED	35.1	NORMAL	34.3	NORMAL	62.5	NORMAL
28	AK	823722		NO RESPONSE	33.7	DECREASED	YES	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE
29	RK	979343	41.7	NORMAL	41.5	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	INCREASED	3.1	NORMAL	13	NORMAL	11.2	NORMAL	53.8	NORMAL
30	RK	843997	34.4	DECREASED	38.8	NORMAL	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.9	NORMAL	2.8	NORMAL	27.7	NORMAL	26.3	NORMAL	61.4	NORMAL
31	LD	848012		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.6	INCREASED	3	NORMAL	9.6	NORMAL	10.8	NORMAL	51.1	NORMAL
32	MK	868747	43.5	NORMAL	48.1	NORMAL	NO	NO	49.6	NORMAL	51.9	NORMAL	DECREASED	NORMAL	2.8	NORMAL	2.5	NORMAL	12.4	NORMAL	17	NORMAL	66	NORMAL
33	BDS	869851	23.9	DECREASED	26.2	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	74.5	INCREASED	DECREASED	NORMAL	4.7	INCREASED	4.7	INCREASED	15.5	NORMAL	12.2	NORMAL	38.3	DECREASED

S.NO	NAME	MRD NO.	(≥40 m/s)		RESPIRATORY INVOLVEMENT			CRANIAL NERVE INVOLVEMENT		NCV FINDINGS		ANY TREATMENT RECEIVED FROM OUTSIDE	TREATMENT RECEIVED IN DMCH LUDHIANA			DISCHARGE STATUS
			RIGHT		RESPIRATORY INVOLVEMENT	TYPE OF RESPIRATORY SUPPORT REQUIRED	NEED FOR MECHANICAL VENTILATION		TYPE		TYPE OF VARIETY		IVIg	I/V STEROIDS	ORAL STEROIDS	
1	AK	620103	64.2	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE MOTOR AXONAL NEUROPATHY	AXONAL	NONE	NO	YES	YES	IMPROVING (DOR)
2	BK	623416	55.6	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING SENSORIMOTOR POLYNEUROPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
3	SS	638200	46.7	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	YES	YES	IMPROVED (DOR)
4	SK	646844		NO RESPONSE	PRESENT	PT ON MECHANICAL VENTILATOR OUTSIDE	YES	YES	BILATERAL 7TH NERVE PALSY	DEMYELINATING SENSORIMOTOR POLYNEURORADICULOPATHY	DEMYELINATING	YES	NO	NO	YES	OPD CASE
5	AK	660069	46.4	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING SENSORIMOTOR POLYNEUROPATHY	DEMYELINATING	NONE	YES	NO	YES	IMPROVING
6	DS	663471	44.3	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
7	R	667314	50.7	NORMAL	ABSENT	NONE	NO	YES	BILATERAL 7TH NERVE PALSY	DEMYELINATING SENSORIMOTOR POLYNEUROPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
8	HS	678065	52.6	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE AXONAL POLYRADICULONEUROPATHY	AXONAL	NONE	YES	NO	NO	IMPROVING
9	PG	677371	48.3	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
10	NK	679377	49	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING SENSORIMOTOR POLYRADICULONEUROPATHY	DEMYELINATING	NONE	NO	NO	NO	OPD CASE
11	MK	698042	60.9	NORMAL	PRESENT	BINASAL OXYGEN SUPPORT	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
12	DK	702439	76.1	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
13	NG	712126	70.7	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE MOTOR AXONAL POLYRADICULONEUROPATHY	AXONAL	NONE	YES	NO	NO	IMPROVED
14	ES	715561	76.1	NORMAL	PRESENT	BINASAL OXYGEN SUPPORT	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVED
15	TL	722336	51.1	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	NO	NO	NO	DAMA
16	HK	726934	71.4	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
17	RB	726888	67.3	NORMAL	ABSENT	NONE	NO	NO	NONE	ACUTE MOTOR AXONAL NEUROPATHY	AXONAL	NONE	NO	NO	NO	IMPROVING
18	JK	727668	52.6	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	AXONAL	NONE	YES	YES	NO	IMPROVING
19	SS	728987	49.6	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
20	S	734436	59.3	NORMAL	PRESENT	TRACHEOSTOMY DONE	YES	YES	BILATERAL 7TH NERVE PALSY	PURE MOTOR AXONAL NEUROPATHY	AXONAL	YES (IVIg)	NO	NO	NO	IMPROVING
21	SS	736396	68.6	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE MOTOR AXONAL NEUROPATHY	AXONAL	NONE	NO	YES	NO	IMPROVED
22	GS	741216	62.5	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE MOTOR AXONAL POLYRADICULONEUROPATHY	AXONAL	NONE	YES	NO	NO	IMPROVING
23	HS	765811	53.8	NORMAL	PRESENT	BINASAL OXYGEN SUPPORT	NO	NO	NONE	DEMYELINATING PREDOMINANTLY MOTOR POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
24	BS	769025		NO RESPONSE	PRESENT	TRACHEOSTOMY DONE	YES	YES	BILATERAL 7TH NERVE PALSY	SENSORY-MOTOR DEMYELINATING POLYNEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
25	PCG	752729	52.2	NORMAL	PRESENT	MECHANICAL VENTILATION	YES	NO	NONE	SENSORI-MOTOR (PREDOMINANT MOTOR) POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	YES	NO	IMPROVING
26	VR	783712	72.9	NORMAL	PRESENT	TRACHEOSTOMY DONE	YES	NO	NONE	PURE MOTOR AXONAL POLYRADICULONEUROPATHY	AXONAL	NONE	YES	YES	NO	IMPROVING
27	SK	803750	58.8	NORMAL	ABSENT	NONE	NO	NO	NONE	BILATERAL C8-T1 RADICULOPATHY (EARLY LGBS)	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
28	AK	823722		NO RESPONSE	PRESENT	MECHANICAL VENTILATION	YES	NO	NONE	DEMYELINATING SENSORIMOTOR POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	YES	YES	IMPROVING
29	RK	979343	56	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
30	RK	843997	60.3	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING POLYRADICULONEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
31	LD	848012	66.7	NORMAL	PRESENT	VENTURI OXYGEN SUPPORT	NO	NO	NONE	DEMEYLINATING POLYRADICULONEUROPATHY	DEMYELINATING	NONE	NO	YES	NO	DAMA
32	MK	868747	74.5	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
33	BDS	869851	37.4	DECREASED	PRESENT	HIGH FLOW OXYGEN SUPPORT	NO	NO	NONE	POLYRADICULONEUROPATHY	DEMYELINATING	NONE	NO	NO	NO	IMPROVING

S.NO	NAME	MRD NO.	AGE (YEARS)	SEX	RESPIRATORY RATE (PER MINUTE) (on admission)	SINGLE BREATH COUNT	POWER (ON ADMISSION)												BILATERAL DEEP TENDON REFLEXES	BILATERAL SENSORY SYSTEM INVOLVEMENT (CLINICAL)	PHR						
							UPPER LIMB						LOWER LIMB								AMPLITUDE (>320MICROVOLT)						
							RIGHT			LEFT			RIGHT			LEFT					MOTOR						
							SHOULDER	ELBOW	HAND GRIP	SHOULDER	ELBOW	HAND GRIP	HIP	KNEE	ANKLE	HIP	KNEE	ANKLE			LEFT		RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LE
34	AA	873937	43	MALE	24	17	0	2	20%	0	2	20%	0	4-	4+	0	4-	4+	ABSENT	NO	NO RESPONSE	NO RESPONSE	42.1	DECREASED	NO	YES	NO RESPONSE
35	KL	874661	61	FEMALE	18	20	3	4	80%	3	4	80%	1	3	4	1	3	4	ABSENT	NO	172.9	DECREASED	164.9	DECREASED	NO	YES	7.5
36	N	879531	20	FEMALE	30	20	2	2	30%	2	2	30%	3	3	3	3	3	3	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
37	AS	882694	18	MALE	24	12	1	1	20%	1	1	20%	3	3	3	3	3	3	ABSENT	NO	672.4	NORMAL	620.5	NORMAL	NO	NO	6.5
38	JS	887234	32	MALE	26	16	1	1	30%	1	1	30%	1	1	1	1	1	1	ABSENT	NO	90.2	DECREASED	68.5	DECREASED	NO	YES	4.2
39	IK	886283	67	FEMALE	18	38	4+	4+	90%	4+	4+	90%	1	3	4-	1	3	4-	ABSENT	NO	190. 4	DECREASED	224.9	DECREASED	NO	YES	2.5
40	MS	119573	78	MALE	16	40	4+	4	90%	4+	4	90%	4	4	4	4	4	4	ABSENT	NO	76.8	DECREASED	89.2	DECREASED	NO	YES	2.2
41	RA	902273	31	MALE	16	14	4+	4-	90%	4+	4+	90%	4+	4+	4	4+	4+	4	ABSENT	NO	440.2	NORMAL	479.7	NORMAL	NO	NO	11
42	JK	907170	24	MALE	18	54	4-	4-	90%	4-	4-	90%	3	3	3	3	3	3	ABSENT	NO	505.8	NORMAL	545.2	NORMAL	NO	NO	8.6
43	SK	906870	64	FEMALE	16	30	4-	3	40%	4-	3	40%	1	1	1	1	1	1	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
44	RK	910170	52	FEMALE	16	20	1	3	90%	1	4	80%	3	2	3	3	2	3	ABSENT	NO	82.2	DECREASED	87.3	DECREASED	NO	YES	8.6
45	SKT	916559	40	MALE	20	20	1	1	20%	1	1	20%	1	1	2	1	1	2	ABSENT	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	NO RESPONSE
46	KS	918168	52	MALE	20	12	3	3	60%	3	3	60%	2	3	4+	2	3	4+	ABSENT	NO	527	NORMAL	592. 8	NORMAL	NO	NO	6.4
47	AS	921637	25	MALE	20	10	2	2	60%	2	2	60%	2	2	2	2	2	2	ABSENT	NO	68.2	DECREASED	49.7	DECREASED	NO	YES	4.8

S.NO	NAME	MRD NO.	ENIC NERVE																							
			ONSET LATENCY (<8.0 MS)					INVOLVED		BILATERAL INVOLVEMENT OF BOTH AMPLITUDE AND LATENCY	MOTOR															VE
											ONSET LATENCY (<4.3 ms)						AMPLITUDE (>5.0 mV)								
			MOTOR								WRIST				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	WRIST				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT			
			FT	RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT	RIGHT		LEFT		RIGHT				LEFT		RIGHT				LEFT		
34	AA	873937	NO RESPONSE	4.8	NORMAL	YES	NO	YES	YES	NO	3.9	NORMAL	3.5	NORMAL	NO	NO	1.2	DECREASED	1.3	DECREASED	NO	YES	53.2	NORMAL	52.1
35	KL	874661	NORMAL	7.9	NORMAL	NO	NO	YES	YES	NO		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
36	N	879531	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES	3.3	NORMAL	3.3	NORMAL	NO	NO	4.5	DECREASED	5.9	NORMAL	YES	NO	57.5	NORMAL	56.8
37	AS	882694	NORMAL	6.6	NORMAL	NO	NO	NO	NO	NO	2.7	NORMAL	3	NORMAL	NO	NO	6.6	NORMAL	8.6	NORMAL	NO	NO	45.5	NORMAL	47.1
38	JS	887234	NORMAL	5.5	NORMAL	NO	NO	YES	YES	NO		NO RESPONSE	2.5	NORMAL	YES	NO		NO RESPONSE	1.2	DECREASED	NO	YES		NO RESPONSE	51.1
39	IK	886283	NORMAL	13.4	INCREASED	YES	NO	YES	YES	NO		NO RESPONSE	10.5	INCREASED	NO	YES		NO RESPONSE	2	DECREASED	NO	YES		NO RESPONSE	54.1
40	MS	119573	NORMAL	2.8	NORMAL	NO	NO	YES	YES	NO	6.8	INCREASED	6.2	INCREASED	NO	YES	5	NORMAL	4.9	DECREASED	YES	NO	40	DECREASED	29.5
41	RA	902273	INCREASED	11.2	INCREASED	NO	YES	YES	YES	NO	5.5	INCREASED	5.9	INCREASED	NO	YES	2.8	DECREASED	1.8	DECREASED	NO	YES	50.5	NORMAL	46.4
42	JK	907170	INCREASED	8.8	INCREASED	NO	YES	YES	YES	NO	3.4	NORMAL	3.3	NORMAL	NO	NO	3.5	DECREASED	9.8	NORMAL	YES	NO	59.7	NORMAL	48.9
43	SK	906870	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
44	RK	910170	INCREASED	8.7	INCREASED	NO	YES	YES	YES	YES	16.1	INCREASED	23.4	INCREASED	NO	YES	1	DECREASED	0.9	DECREASED	NO	YES	49.5	NORMAL	41.1
45	SKT	916559	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO	YES	YES	YES	YES	3.1	NORMAL	3.3	NORMAL	NO	NO	0.6	DECREASED	0.7	DECREASED	NO	YES	50	NORMAL	53.5
46	KS	918168	NORMAL	6.4	NORMAL	NO	NO	NO	NO	NO	3.8	NORMAL	3.6	NORMAL	NO	NO	6.4	NORMAL	6.2	NORMAL	NO	NO	51.6	NORMAL	56.5
47	AS	921637	NORMAL	5.7	NORMAL	NO	NO	YES	YES	NO	3.3	NORMAL	3.1	NORMAL	NO	NO	6.7	NORMAL	5.9	NORMAL	NO	NO	51.1	NORMAL	55.4

S.NO	NAME	MRD NO.	UPPER LIMB																									
			MEDIAN NERVE																									
			LOCITY (>45 m/s)			F- WAVE LATENCY (<31.2 ms)				F- WAVE PERSISTENCE (>50%)		SENSORY														ONSET LAT		
												PEAK LATENCY (<3.6 ms)				AMPLITUDE (>11 microV)				VELOCITY (≥50 m/s)				WRIST		
			RIGHT	U/L INVOLVEME	B/L INVOLVEME	LEFT		RIGHT		LEFT	RIGHT	LEFT		RIGHT		LEFT		RIGHT		LEFT		RIGHT		LEFT		RI
34	AA	873937	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.2	NORMAL	2.9	NORMAL	52.1	NORMAL	40.1	NORMAL	56.9	NORMAL	63.6	NORMAL	3.8	INCREASED	2.8
35	KL	874661	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	2.7	NORMAL	2.5
36	N	879531	NORMAL	NO	NO	25.4	NORMAL	25.3	NORMAL	DECREASED	DECREASED	2.5	NORMAL	2.9	NORMAL	28.5	NORMAL	35.3	NORMAL	72.2	NORMAL	68.6	NORMAL	2.8	NORMAL	2.5
37	AS	882694	NORMAL	NO	NO	27.2	NORMAL	25.2	NORMAL	DECREASED	DECREASED	2.6	NORMAL	3	NORMAL	45	NORMAL	46.8	NORMAL	68	NORMAL	63.1	NORMAL	2	NORMAL	2.2
38	JS	887234	NORMAL	YES	NO	NOT RECORDED	NOT RECORDED	28.7	NORMAL	NOT RECORDED	NORMAL	2.9	NORMAL	2.6	NORMAL	54	NORMAL	38.3	NORMAL	63.1	NORMAL	64.2	NORMAL		NO RESPONSE	
39	IK	886283	NORMAL	YES	NO	NOT RECORDED	NOT RECORDED	41.1	INCREASED	NOT RECORDED	NORMAL	4	INCREASED		NO RESPONSE	5.1	DECREASED		NO RESPONSE	51.9	NORMAL		NO RESPONSE	2.6	NORMAL	2.9
40	MS	119573	DECREASED	NO	YES	39.9	INCREASED	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	5.6	INCREASED	5.8
41	RA	902273	NORMAL	NO	NO	32.6	INCREASED	35.3	INCREASED	DECREASED	DECREASED	4.3	INCREASED	4.3	INCREASED	9.4	DECREASED	13.2	NORMAL	41.9	DECREASED	45.5	DECREASED	3	NORMAL	2.7
42	JK	907170	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	3	NORMAL	31.4	NORMAL	30.7	NORMAL	67.3	NORMAL	61.4	NORMAL	2.5	NORMAL	2.7
43	SK	906870	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	
44	RK	910170	DECREASED	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	6.2	INCREASED	6.8
45	SKT	916559	NORMAL	NO	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.9	NORMAL	2.8	NORMAL	42.6	NORMAL	40.6	NORMAL	62.5	NORMAL	70.7	NORMAL		NO RESPONSE	
46	KS	918168	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	29.5	NORMAL	DECREASED	DECREASED	3.9	INCREASED	3.6	NO	44.7	NORMAL	46.3	NORMAL	49	DECREASED	52.2	NORMAL	3.4	NORMAL	3
47	AS	921637	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	2.6	NORMAL	54.8	NORMAL	55.1	NORMAL	69.3	NORMAL	77.8	NORMAL	4	INCREASED	3.1

S.NO	NAME	MRD NO.	ULNAR NERVE																									
			MOTOR																F- WAVE LATENCY (<32.1 ms)				F- WAVE PERSISTENCE (>50%)		SENSORY			
			ENCY (<3.5 ms)			AMPLITUDE (>5.5 mV)						VELOCITY (>43 m/s)													PEAK LATENCY (<3.0 ms)		
			GHT	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	WRIST				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT		RIGHT		U/L INVOLVEME	B/L INVOLVEME	LEFT		RIGHT		LEFT	RIGHT	LEFT		RI
34	AA	873937	NORMAL	YES	NO	2.4	DECREASED	1.3	DECREASED	NO	YES	72.7	NORMAL	55.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	2.6
35	KL	874661	NORMAL	NO	NO	3.8	DECREASED	5.6	NORMAL	YES	NO	39	DECREASED	50	NORMAL	YES	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	3
36	N	879531	NORMAL	NO	NO	1.5	DECREASED	3.6	DECREASED	NO	YES	87.3	NORMAL	65.8	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.3	NORMAL	2.7
37	AS	882694	NORMAL	NO	NO	9.6	NORMAL	7.2	NORMAL	NO	NO	50.6	NORMAL	51.2	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	25.6	NORMAL	DECREASED	DECREASED	2.5	NORMAL	2.6
38	JS	887234	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.6	NORMAL	2.6
39	IK	886283	NORMAL	NO	NO	7.2	NORMAL	6.4	NORMAL	NO	NO	64.9	NORMAL	57.8	NORMAL	NO	NO	29.9	NORMAL	31.9	NORMAL	NORMAL	NORMAL	3.1	INCREASED	2.8
40	MS	119573	INCREASED	NO	YES	2.5	DECREASED	5.7	NORMAL	YES	NO	24.2	DECREASED	25	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE	
41	RA	902273	NORMAL	NO	NO	6.1	NORMAL	4.9	DECREASED	YES	NO	62.8	NORMAL	48.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	31.9	NORMAL	DECREASED	NORMAL	3.2	INCREASED	3.3
42	JK	907170	NORMAL	NO	NO	6.8	NORMAL	8	NORMAL	NO	NO	63.2	NORMAL	71.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.7	NORMAL	2.7
43	SK	906870	NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE	
44	RK	910170	INCREASED	NO	YES	1.1	DECREASED	1.4	DECREASED	NO	YES	57.1	NORMAL	44.4	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED		NO RESPONSE	
45	SKT	916559	NO RESPONSE	NO	NO		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.8	NORMAL	2.8
46	KS	918168	NORMAL	NO	NO	5.6	NORMAL	7	NORMAL	NO	NO	58.8	NORMAL	58.1	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3	NORMAL	3.5
47	AS	921637	NORMAL	YES	NO	2.1	DECREASED	2.6	DECREASED	NO	YES	73.8	NORMAL	69.6	NORMAL	NO	NO	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	2.5	NORMAL	2.8

S.NO	NAME	MRD NO.	SENSORY									MOTOR											PERONEAL NERVE			
				AMPLITUDE (>11 microV)				VELOCITY (≥50 m/s)				ONSET LATENCY (<5.4 ms)						AMPLITUDE (>2.0 mV)								VEL
												ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	ANKLE				UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT			
			GHT	LEFT		RIGHT		LEFT		RIGHT		LEFT		RIGHT				LEFT		RIGHT				LEFT		F
34	AA	873937	NORMAL	39.5	NORMAL	41.9	NORMAL	65.4	NORMAL	76.1	NORMAL	7	INCREASED	6.1	INCREASED	NO	YES	0.7	DECREASED	0.9	DECREASED	NO	YES	48	NORMAL	45.5
35	KL	874661	NORMAL	44	NORMAL	33.7	NORMAL	61.4	NORMAL	60.9	NORMAL		NO RESPONSE	4.2	NORMAL	YES	NO		NO RESPONSE	2.4	NORMAL	YES	NO		NO RESPONSE	31.7
36	N	879531	NORMAL	32.1	NORMAL	27.5	NORMAL	80.5	NORMAL	63.6	NORMAL	3.7	NORMAL	4.6	NORMAL	NO	NO	1.5	DECREASED	1.1	DECREASED	NO	YES	33	DECREASED	34.5
37	AS	882694	NORMAL	38	NORMAL	31.8	NORMAL	76.1	NORMAL	68	NORMAL	4.1	NORMAL	4	NORMAL	NO	NO	4.3	NORMAL	3.8	NORMAL	NO	NO	36.5	DECREASED	31.6
38	JS	887234	NORMAL	40.9	NORMAL	44.7	NORMAL	76.9	NORMAL	71.4	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
39	IK	886283	NORMAL	30.4	NORMAL	5.2	DECREASED	61.9	NORMAL	102.9	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
40	MS	119573	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	10	INCREASED		NO RESPONSE	NO	YES	1.1	DECREASED		NO RESPONSE	NO	YES	19.3	DECREASED	
41	RA	902273	INCREASED	31.8	NORMAL	35.9	NORMAL	55.6	NORMAL	50.7	NORMAL	6.5	INCREASED	5.2	NORMAL	YES	NO	1.4	DECREASED	1	DECREASED	NO	YES	25.2	DECREASED	31.1
42	JK	907170	NORMAL	24.9	NORMAL	40.1	NORMAL	75.3	NORMAL	71.4	NORMAL	5.8	INCREASED	6.7	INCREASED	NO	YES	8.6	NORMAL	7.3	NORMAL	NO	NO	41.9	NORMAL	42.5
43	SK	906870	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
44	RK	910170	NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE	11.3	INCREASED	8.2	INCREASED	NO	YES	0.6	DECREASED	0.9	DECREASED	NO	YES	38	NORMAL	36.1
45	SKT	916559	NORMAL	29.1	NORMAL	30.1	NORMAL	69.3	NORMAL	70.7	NORMAL		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE	
46	KS	918168	INCREASED	26.5	NORMAL	29.3	NORMAL	34.5	DECREASED	54.3	NORMAL	5.3	NORMAL	5	NORMAL	NO	NO	6.8	NORMAL	3.4	NORMAL	NO	NO	43.5	NORMAL	41.3
47	AS	921637	NORMAL	41	NORMAL	32.3	NORMAL	78.7	NORMAL	72.2	NORMAL		NO RESPONSE	5.9	INCREASED	NO	YES		NO RESPONSE	1.3	DECREASED	NO	YES		NO RESPONSE	35.3

S.NO	NAME	MRD NO.	LOWER LIMB																					
													TIBIAL NER											
													MOTOR											
			OCITY (≥37 m/s)			F- WAVE LATENCY (<59.3 ms)				F- WAVE PERSISTENCE (>30%)			ONSET LATENCY (<6.1 ms)						AMPLITUDE (>3.5 mV)					
													ANKLE						ANKLE					
			IGHT	U/L INVOLVEMENT	B/L INVOLVEMENT	LEFT		RIGHT		LEFT	RIGHT		LEFT		RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	LEFT		RIGHT		UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT
34	AA	873937	NORMAL	NO	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	7.9	INCREASED	6.1	NORMAL	YES	NO	0.8	DECREASED	0.5	DECREASED	NO	YES
35	KL	874661	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NO RESPONSE	NO RESPONSE	NOT RECORDED	DECREASED	4.3	NORMAL	4.6	NORMAL	NO	NO	1.5	DECREASED	3.8	NORMAL	YES	NO
36	N	879531	DECREASED	NO	YES	50.6	NORMAL	50.5	NORMAL	DECREASED	NORMAL	3.7	NORMAL	4.1	NORMAL	NO	NO	5.6	NORMAL	5.7	NORMAL	NO	NO
37	AS	882694	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	27.5	NORMAL	DECREASED	NORMAL	4.7	NORMAL	4.7	NORMAL	NO	NO	11.7	NORMAL	10.5	NORMAL	NO	NO
38	JS	887234	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	4.8	NORMAL	4.9	NORMAL	NO	NO	1.2	DECREASED	0.9	DECREASED	NO	YES
39	IK	886283	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	7.4	INCREASED	12	INCREASED	NO	YES	1	DECREASED	1.2	DECREASED	NO	YES
40	MS	119573	NO RESPONSE	NO	YES	NO RESPONSE	NO RESPONSE	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	13.3	INCREASED	9.9	INCREASED	NO	YES	1.8	DECREASED	2.5	DECREASED	NO	YES
41	RA	902273	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	6.9	INCREASED	4.6	NORMAL	YES	NO	3.4	DECREASED	3.5	NORMAL	YES	NO
42	JK	907170	NORMAL	NO	NO	49.7	NORMAL	52.5	NORMAL	DECREASED	DECREASED	4.6	NORMAL	5	NORMAL	NO	NO	6.7	NORMAL	6.7	NORMAL	NO	NO
43	SK	906870	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
44	RK	910170	DECREASED	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
45	SKT	916559	NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE	NO	YES		NO RESPONSE		NO RESPONSE	NO	YES
46	KS	918168	NORMAL	NO	NO	57.8	NORMAL	49.5	NORMAL	NORMAL	DECREASED	5.2	NORMAL	4.2	NORMAL	NO	NO	11.3	NORMAL	12.2	NORMAL	NO	NO
47	AS	921637	DECREASED	NO	YES	NOT RECORDED	NOT RECORDED	NO RESPONSE	NO RESPONSE	NOT RECORDED	DECREASED	6	NORMAL	5.4	NORMAL	NO	NO	0.8	DECREASED	0.7	DECREASED	NO	YES

S.NO	NAME	MRD NO.	VE												SURAL NERVE									
			VELOCITY (>35 m/s)						F- WAVE LATENCY (<54 ms)				F- WAVE PERSISTENCE (>50%)		SENSORY									
															PEAK LATENCY (<3.1 ms)				AMPLITUDE (>9.0 microV)				VELOCITY	
			LEFT		RIGHT		U/L INVOLVEMENT	B/L INVOLVEMENT	LEFT		RIGHT		LEFT	RIGHT	LEFT		RIGHT		LEFT		RIGHT		LEFT	
34	AA	873937	46.4	NORMAL	46.4	NORMAL	NO	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	2.9	NORMAL	2.9	NORMAL	31.7	NORMAL	26.5	NORMAL	64.2	NORMAL
35	KL	874661	30	DECREASED	30.8	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	4.1	INCREASED	3.4	INCREASED	11.9	NORMAL	14.1	NORMAL	45.2	NORMAL
36	N	879531	36	NORMAL	34.4	DECREASED	YES	NO	51.5	NORMAL	56.3	INCREASED	NORMAL	DECREASED	3.2	INCREASED	3.1	NORMAL	13.6	NORMAL	12.7	NORMAL	58.3	NORMAL
37	AS	882694	38.9	NORMAL	32.4	DECREASED	YES	NO	NO RESPONSE	NO RESPONSE	37.1	NORMAL	DECREASED	NORMAL	3.7	INCREASED	4.3	INCREASED	37.3	NORMAL	30.2	NORMAL	48.3	NORMAL
38	JS	887234	36	NORMAL	43	NORMAL	NO	NO	49.5	NORMAL	NOT RECORDED	NOT RECORDED	DECREASED	NOT RECORDED	2.9	NORMAL	3	NORMAL	28.8	NORMAL	26.9	NORMAL	63.1	NORMAL
39	IK	886283	26.2	DECREASED	27.8	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	NO RESPONSE	NO RESPONSE	DECREASED	DECREASED	3.9	INCREASED	4	INCREASED	12.3	NORMAL	13.6	NORMAL	48.6	NORMAL
40	MS	119573	21	DECREASED	16.6	DECREASED	NO	YES	NO RESPONSE	NO RESPONSE	60.7	INCREASED	DECREASED	NORMAL		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE
41	RA	902273	31.1	DECREASED	29.4	DECREASED	NO	YES	62.3	INCREASED	53.1	NORMAL	NORMAL	NORMAL	3.9	INCREASED	4	INCREASED	24	NORMAL	20.7	NORMAL	46.4	NORMAL
42	JK	907170	33.6	DECREASED	34.6	DECREASED	NO	YES	60.9	INCREASED	56.1	INCREASED	DECREASED	NORMAL	3.7	NORMAL	3.3	NORMAL	22.8	NORMAL	20.4	NORMAL	52.2	NORMAL
43	SK	906870		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE		NO RESPONSE
44	RK	910170		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.3	INCREASED	3.3	INCREASED	17.3	NORMAL	12.3	NORMAL	58.8	NORMAL
45	SKT	916559		NO RESPONSE		NO RESPONSE	NO	YES	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.6	INCREASED	3.7	INCREASED	35.2	NORMAL	37.5	NORMAL	52.6	NORMAL
46	KS	918168	42.8	NORMAL	34.2	NORMAL	NO	NO	53.1	NORMAL	38,5	NORMAL	DECREASED	NORMAL	3.5	INCREASED	1.9	NORMAL	29.7	NORMAL	33	NORMAL	58.3	NORMAL
47	AS	921637	39	NORMAL	31.1	DECREASED	YES	NO	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	NOT RECORDED	3.5	INCREASED	3.1	NORMAL	29.7	NORMAL	34.6	NORMAL	53	NORMAL

S.NO	NAME	MRD NO.	(≥40 m/s)		RESPIRATORY INVOLVEMENT			CRANIAL NERVE INVOLVEMENT		NCV FINDINGS		ANY TREATMENT RECEIVED FROM OUTSIDE	TREATMENT RECEIVED IN DMCH LUDHIANA			DISCHARGE STATUS
			RIGHT		RESPIRATORY INVOLVEMENT	TYPE OF RESPIRATORY SUPPORT REQUIRED	NEED FOR MECHANICAL VENTILATION		TYPE		TYPE OF VARIETY		IVIg	I/V STEROIDS	ORAL STEROIDS	
34	AA	873937	64.2	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	AXONAL	NONE	YES	YES	YES	IMPROVING
35	KL	874661	54.7	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULONEUROPATHY	AXONAL	NONE	YES	YES	YES	IMPROVING
36	N	879531	59.8	NORMAL	PRESENT	MECHANICAL VENTILATION	YES	NO	NONE	POLYRADICULOPATHY	AXONAL	NONE	NO	YES	YES	DAMA
37	AS	882694	41.9	NORMAL	PRESENT	MECHANICAL VENTILATION	YES	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	YES	YES	DAMA
38	JS	887234	63.1	NORMAL	PRESENT	TRACHEOSTOMY DONE	YES	YES	BILATERAL 7TH NERVE PALSY; BULBAR PALSY +	PURE MOTOR AXONAL NEUROPATHY	AXONAL	YES (IVIg)	NO	YES	NO	IMPROVING
39	IK	886283	44.3	NORMAL	ABSENT	NONE	NO	YES	RIGHT SIDE 7TH NERVE PALSY	AXONAL SENSORIMOTOR POLYRADICULONEUROPATHY	AXONAL	NONE	YES	NO	NO	IMPROVING
40	MS	119573		NO RESPONSE	ABSENT	NONE	NO	NO	NONE	DEMYELINATING SENSORIMOTOR POLYRADICULONEUROPATHY	DEMYELINATING	NONE	NO	YES	YES	IMPROVING
41	RA	902273	47.9	NORMAL	ABSENT	NONE	NO	YES	RIGHT 7TH NERVE PALSY	POLYRADICULONEUROPATHY	DEMYELINATING	NONE	NO	NO	NO	IMPROVING
42	JK	907170	57.4	NORMAL	ABSENT	NONE	NO	NO	NONE	POLYRADICULOPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVED
43	SK	906870		NO RESPONSE	ABSENT	NONE	NO	NO	NONE	SENSORY-MOTOR POLYNEUROPATHY	COULD NOT BE ASSESSED	NONE	YES	NO	NO	IMPROVED
44	RK	910170	58.8	NORMAL	ABSENT	NONE	NO	NO	NONE	DEMYELINATING SENSORIMOTOR POLYNEUROPATHY	DEMYELINATING	NONE	YES	NO	NO	IMPROVING
45	SKT	916559	49.6	NORMAL	ABSENT	NONE	NO	NO	NONE	PURE MOTOR AXONAL NEUROPATHY	AXONAL	NONE	YES	NO	NO	IMPROVING
46	KS	918168	50.7	NORMAL	ABSENT	NONE	NO	NO	NONE	EARLY LGBS	DEMYELINATING	NONE	YES	YES	NO	IMPROVING
47	AS	921637	57.9	NORMAL	ABSENT	NONE	NO	NO	NONE	ACUTE MOTOR AXONAL NEUROPATHY	AXONAL	NONE	YES	NO	YES	IMPROVING

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Date 9/9/19

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Anchal Aggarwal**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Phrenic nerve conduction abnormalities in landry guillain barre syndrome

Supervisor
Dr. Rajesh Mahajan
Prof.,
Dept. of Medicine,
DMCH, Ludhiana.

Co-Supervisor
Dr. Rajinder K. Bansal
Prof.,
Dept. of Neurology,
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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

SP Singh
Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

IN

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

October, 2018

DR. ANCHAL AGGARWAL

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE & HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	Phrenic nerve conduction abnormalities in Landry Guillain Barre Syndrome.
For the degree of	MD MEDICINE
Name of the candidate	Dr. Anchal Aggarwal
Supervisor	Dr. Rajesh Mahajan, Professor, Department of Medicine, DMCH, Ludhiana
Co-supervisor	Dr. Rajinder K. Bansal, Professor, Department of Neurology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

INTRODUCTION: : Landry Guillain Barre Strohl Syndrome is an acute fulminant polyradiculoneuropathy that is autoimmune in nature with infectious/ non-infectious triggers occurring annually at a rate of 1-4 cases per 1,00,000. It is one of the most common cause of neuromuscular respiratory failure with 20-30 % patients requiring mechanical ventilation as respiratory failure contributes to significant morbidity and mortality in these patients.

Aim of Study: To study various phrenic nerve conduction abnormalities and to see if they can predict impending respiratory failure in LGBS patients.

Materials & Methods: This will be a prospective study conducted on patients of age ≥ 18 years who present to DMCH Ludhiana with rapidly evolving areflexic ascending paralysis or quadriparesis or having the suspicion of having LGBS and NCV studies including Phrenic Nerve will be done and patients will be followed up for development of any respiratory abnormalities during hospital stay

Clinical Significance: : This study will help us in documenting the various abnormalities in Phrenic Nerve conduction studies and their role in predicting any respiratory involvement in LGBS patients in order to individualise timely management in such patients as respiratory distress is the most important cause of death in GBS in acute phase.

Keywords: Landry Guillain Barre Syndrome, NCV, Phrenic Nerve, Respiratory involvement.

(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis entitled **"PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME "** exist in the Department of Medicine, Dayanand Medical College & Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Rajesh Mahajan
Professor
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Co-Supervisor(s)

Dr. Rajinder K Bansal
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Place : LUDHIANA

Date : 01-11-2018

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Anchal Aggarwal
Department	MEDICINE
Topic of Thesis	Phrenic nerve conduction abnormalities in Landry Guillain Barre Syndrome
Likely date of appearing in PG examination	JUNE 2021
Date of enrollment	9th MAY 2018
Name of Head of Department	Dr. Dinesh Gupta
1) Supervisor	Dr. Rajesh Mahajan
2) Co-Supervisor	Dr. Rajinder K Bansal
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. & Gynae.
	8. Legal Expert
	9. Secretary, IEC
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	11. Vice Principal
	12. Principal

Approved: (Yes) / No

Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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2	NAME OF THE INSTITUTE	Dayanand Medical College and Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	MD MEDICINE
4	DATE OF ADMISSION OF COURSE	9th May, 2018
5	TITLE OF THE TOPIC	PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME.

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Landry Guillain Barre Srohl Syndrome is an acute acquired fulminant polyradiculoneuropathy that is autoimmune in nature with GM1 antibodies responsible for AIDP and GQ1b antibodies responsible for Miller Fisher syndrome, triggered by infectious or non-infectious agents presenting with progressively flaccid, symmetric and ascending weakness of extremities. Legs are usually more commonly involved than arms. The lower cranial nerves are also frequently involved causing bulbar weakness with difficulty handling secretions and maintaining airways. Most patients require hospitalization and upto 30 % require respiratory assistance at some time during the course of illness. The need for mechanical ventilation is associated with more severe weakness on admission, a rapid tempo of progression, and the presence of facial and/or bulbar weakness during the first week of symptoms. Deep tendon reflexes attenuate or disappear within the first few days of onset. Cutaneous sensory deficits (e.g., loss of pain and temperature sensation) are usually relatively mild, but functions subserved by large sensory fibers, such as deep tendon reflexes and proprioception, are more severely affected. Bladder dysfunction may occur in severe cases but is usually transient. Several subtypes of GBS are recognized, as determined primarily by electrodiagnostic and pathologic distinctions.

1. Acute inflammatory demyelinating polyneuropathy (AIDP)
2. Acute motor axonal neuropathy (AMAN)
3. Acute motor sensory axonal neuropathy (AMSAN)
4. Miller fisher syndrome (MFS)

Approximately 70% of cases of GBS occur 1–3 weeks after an acute infectious process, usually respiratory or gastrointestinal especially with Campylobacter jejuni infection. Other antecedent infections associated with GBS are HHV infection like EBV and CMV, HIV, hepatitis E and Mycoplasma pneumonia.

It is one of the most common causes of neuromuscular respiratory failure with 20-30 % patients requiring mechanical ventilation. Upon presentation, 40 % of patients have respiratory or oropharyngeal weakness with typical complaints of dyspnea on exertion, shortness of breath, difficulty swallowing and slurred speech. Ventilatory failure which requires respiratory support occurs in upto 1/3 of patients at sometime during the course of their disease. So, early prediction of mechanical ventilation in GBS patients is of great importance to enable physicians individualize supportive treatment. As respiratory failure contributes to significant morbidity and mortality in patients with GBS, monitoring of lung functions, anticipation of mechanical ventilation and pulmonary rehabilitation are important for optimal treatment and recovery of these patients. Regular monitoring of respiratory function (vital capacity) is needed in the acute phase, as respiratory failure may develop with little warning. Many clinical, pulmonary and electrophysiological parameters have been extensively used to predict respiratory failure in GBS patients, as timely ventilation may change the course and prognosis for the patient. Using clinical and pulmonary parameters may give inter-observer errors but using electrophysiological studies especially for phrenic nerve, can reduce such errors and might aid in predicting the onset of respiratory problems in these patients.

In the demyelinating forms of GBS, the basis for flaccid paralysis and sensory disturbance is conduction block. This finding, demonstrable electrophysiologically, implies that the axonal connections remain intact. Hence, recovery can take place rapidly as remyelination occurs.

Diagnosis of LGBS: Brighton criteria is used

1. Clinical suspicion
2. CSF analysis suggestive of albumino-cytological dissociation
3. Electrodiagnostic studies (NCV)

IMPORTANCE OF ELECTRODIAGNOSTIC STUDIES IN LGBS:

Electrodiagnostic studies are performed to support the clinical impression which demonstrate features of demyelination, such as temporal dispersion and conduction block, significantly slow conduction velocities, and prolonged distal and F-wave latencies. These are particularly helpful because these findings are characteristic of immune-mediated demyelinating neuropathies.

In GBS, the inexcitability of nerves leading to flaccid paralysis and sensory disturbance is due to severe conduction blocks or axonal pathology which is the basis for analyzing the data using compound muscle action potential amplitudes.

Treatment of LGBS: Plasmapheresis and intravenous immunoglobulis are the two main immunotherapy treatments for GBS. Respiratory involvement may require intubation of trachea and breathing support through mechanical ventilation generally in ICU setting.

The need for ventilation can be anticipated by the following bedside parameters:

i) Forced vital capacity (FVC)

ii) Single breath count

iii) Time taken to reach maximum deficit

iv) Neck weakness

v) Facial weakness

vi) Bulbar weakness

vii) Phrenic nerve conduction studies

6.2 REVIEW OF LITERATURE

Guillain Barre Syndrome is the most common and most severe acute paralytic neuropathy. Guillain Barre Syndrome is a clinically suspected diagnosis but nerve conduction studies can help support diagnosis to discriminate between axonal and demyelinating subtypes and could relate to prognosis.

Mortality in GBS due to ventilatory failure is around 20 %. Early recognition and timely institution of appropriate measures result in considerable reduction of mortality. Clinical assessment and vital capacity measurement though useful are not sensitive enough to detect ventilatory failure in early stages. Certain studies also showed that phrenic nerve conduction studies provided the earliest indication of involvement of respiratory muscles and in most instances heralded ventilatory insufficiency.

In a study by Barun Kumar Sen and Alak Pandit [1], abnormal phrenic nerve conduction studies were seen in 65.62 % cases (42 out of 64 patients) and among these cases, 45.24 % (19 out of 42 patients) developed respiratory failure. 7 out of 19 patients on admission had respiratory failure and 12 out of 19 developed respiratory failure during hospital stay. They also found that Single Breath Count was significantly low in those patients who developed respiratory failure. With normal phrenic nerve study, no one developed respiratory failure (NPV- 100%) and with abnormal phrenic nerve conduction study, almost half patients developed respiratory failure and all those who developed respiratory failure had abnormal phrenic nerve study (sensitivity- 100%). Thus patients with GBS with normal phrenic nerve conduction study are unlikely to develop significant respiratory failure. Hence phrenic nerve conduction study may be considered independently in predicting respiratory

failure. In phrenic nerve conduction studies they showed that its sum latency, amplitude, duration and area were abnormal in those who developed respiratory failure.

Clinical assessment and vital capacity measurements, though useful, are not sensitive enough to detect ventilation failure in the early stages as found in Ito *et al.*'s study [2] where clinical assessment was not correlated with phrenic nerve conduction or subsequent respiratory failure, but phrenic nerve conduction studies were proportional to measurements of respiratory functions such as vital capacity and provided the earliest indicator of involvement of respiratory muscles.[3-4]

Davis [5] described a simple surface recording method of phrenic nerve conduction study in humans and confirmed that it represented the true diaphragmatic muscle action potential. According to their study, clinical assessment and vital capacity measurement though useful, are not sensitive enough to detect ventilatory failure in the early stages Theoretically, phrenic nerve conduction studies could be used as a marker of respiratory muscle weakness.

In a study by Gourie-Devi and Ganapathy [6] abnormalities in phrenic nerve conduction time were observed in 18 out of 28 patients (64.3%) at admission or on subsequent examination and 83.3% of patients with prolonged phrenic nerve conduction time developed ventilatory failure during the course of the illness. Conduction time was found to be prolonged in 18 (64.3%) patients and serial studies showed progressive improvement with restoration of normal values in majority by 12 weeks. These conduction abnormalities had a positive correlation with the extent of the disease, morbidity and mortality.

Phrenic nerve conduction time was found to be more sensitive parameter than vital capacity or median nerve motor conduction velocity in assessing the severity of the disease and predicting impending ventilatory failure.

Early progression to peak disability, bulbar dysfunction and autonomic instability predicted the development of neuromuscular respiratory paralysis in GBS. Early electrodiagnostic studies in this series suggest axonopathic GBS as a predictor of respiratory paralysis.[7]

Another study by Basiri et al [8] showed that compound muscle action potential (CMAP) latencies, right diaphragmatic CMAP amplitude and diaphragmatic CMAP duration were significantly different between GBS patients with respiratory failure and without respiratory failure concluding that not only phrenic nerve CMAP latency and amplitude but also diaphragmatic CMAP duration could have predictive value for respiratory failure in GBS patients.

Study by U Zifco et al [9] indicate that involvement of phrenic nerve and diaphragm is common at an early stage of disease, occurring in 88 % of patients. Furthermore, some respiratory electrophysiological variables are sensitive predictors of severe respiratory failure and are superior to spirometry in terms of early recognition of patients who are at risk of severe diaphragmatic weakness. Their study also showed that prolonged phrenic nerve latency was the most common finding (78%), being present in ventilated as well as non-ventilated patients suggesting that either axonal degeneration or mild segmental demyelination is occurring in early course of disease. Although phrenic nerve conduction and needle EMG of the diaphragm showed a sensitivity of 100 % in predicting respiratory failure but they were not specific. Patients without respiratory failure had abnormal

phrenic nerve latencies (79%), abnormal diaphragmatic CMAP amplitudes (29%) and abnormal findings on needle EMG of the diaphragm (29%). However, no patient with normal electrophysiological respiratory studies developed respiratory failure. Thus patients with GBS and normal phrenic nerve conduction and normal diaphragmatic needle EMG are unlikely to develop significant ventilatory failure.

Increasing studies [10-16] have focused on clinical predictors to assess need for mechanical ventilation in GBS patients including time from onset to admission of < 7 days, inability to cough and stand, inability to lift elbows or head from the bed, cranial nerve deficits, increased liver enzymes, anti-GQ1b antibodies, low vital capacity, phrenic nerve CMAP latency and the proximal/ distal compound muscular amplitude potential (p/d CMAP) ratio of the peroneal nerve. Early prediction of mechanical ventilation is important in that 60 % of patients with mechanical ventilation developed major complications including pneumonia, sepsis, gastrointestinal bleeding. So early identification and intervention may reduce risk of complications and improve the outcome of patients. A study by Xiujuan Wu et al [17] revealed that shorter interval from onset to admission, facial nerve palsy, glossopharyngeal and vagus nerve deficits and lower MRC sum score were risk factors for mechanical ventilation.

According to a study by Hugh J Willison et al [18], importance of NCV is:

• Can be helpful in clinical practice, but are generally not required to diagnose Guillain-Barré syndrome.

• Needed to meet all Brighton criteria for Guillain-Barré syndrome.[19]

• Essential for classification of Guillain-Barré syndrome in acute inflammatory demyelinating polyneuropathy or acute motor axonal neuropathy.

• Acute inflammatory demyelinating polyneuropathy: features of demyelination (decreased motor nerve conduction velocity, prolonged distal motor latency, increased F-wave latency, conduction blocks, and temporal dispersion).[20]

• Acute motor axonal neuropathy: no features of demyelination (one demyelinating feature in one nerve, if distal CMAP amplitude is less than 10% LLN, can be found; distal CMAP amplitude less than 80% LLN in at least two nerves.[20] Transient motor nerve conduction block might be present.[21]

In our study we are going to assess the importance of phrenic nerve conduction studies in predicting respiratory abnormalities and the various abnormalities observed in phrenic nerve conduction studies.

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To assess involvement of Phrenic nerve using non- invasive Phrenic nerve conduction studies in LGBS patients.
2. To study various types of conduction abnormalities in Phrenic Nerve electrophysiological studies in LGBS patients.
3. To see if Phrenic Nerve conduction abnormalities have a role in predicting onset of impending respiratory failure in LGBS patients.

7. MATERIALS AND METHODS

All patients admitted to DMCH Ludhiana under Department of Medicine and Department of Neurology from 1st November 2018 to 30th April 2020 fulfilling the inclusion criteria and not falling in the exclusion criteria, and giving consent for being enrolled in our study will be included.

All patients admitted to DMCH Ludhiana with suspicion of having LGBS with complaints of :

- leg and arm weakness (32%) or
- selective proximal and distal leg weakness (56%) often spreading to the arm or
- weakness in arm or
- symmetric ascending paralysis or
- paraparesis or
- quadriparesis or
- difficulty bearing weight on both legs or
- Acroparesthesia

who will be suspected to have GBS will be taken into study after written consent and NCV studies including Phrenic Nerve studies will be performed on them.

Definition of Single Breath Count (SBC): Measurement of how far an individual can count in a normal speaking voice after a maximal effort inhalation.

<u>DETAILS OF NCV STUDIES TO BE PERFORMED:</u>

Active electrodes are placed on the center of muscle belly with reference electrodes being placed over muscle tendon distally. Ground electrodes are

also placed between active and reference electrode. Stimulator for stimulating the nerve under study is placed over the nerve that supplies the muscle. It has a cathode (negative pole) which depolarizes the underlying nerve segment and an anode (positive pole) which hyperpolarizes underlying nerve segment. As the nerve is stimulated, a compound muscle action potential (CMAP) is generated which represents the summation of all underlying individual muscle fibre action potentials. The following parameters of this muscle action will be studied:

- Latency: time interval between onset of stimulus and onset of response
- Amplitude: maximum height of action potential
- Conduction Velocity: It is the speed of propagation of an electrical impulse through the nerve calculated as distance travelled by the impulse per unit time.
- F-wave latency: It is the shortest latency to onset of the initial deflection (either negative or positive) of all recorded F waves which are compound action potentials evoked by supra-maximal antidromic stimulation of a motor nerve.
- F-wave persistence: The number of occurrences of response divided by number of stimuli which is a measure of antidromic excitability of a particular motor neuron pool.

ELECTRODES PLACEMENT

			RECORDING ELECTRODES		STIMULATION SITES
			ACTIVE ELECTRODE (G1)	REFERENCE ELECTRODE (G2)	
UPPER LIMB	MEDIAN NERVE	MOTOR	Abductor pollicis brevis muscle belly	First metacarpo-phalangeal joint	1. Middle of wrist between tendons of FCR and Palmaris longus 2. Antecubital fossa over brachial artery pulse
		SENSORY	Index/middle finger Metacarpo-phalangeal joint	3-4 cm distally over DIP joint	Middle of wrist between tendons of FCR and Palmaris longus
	ULNAR NARVE	MOTOR	Abductor digiti minimi muscle belly	Fifth metacarpo-phalangeal joint	1.Medial wrist adjacent to FCU tendon 2. Elbow 3cm distal to medial epicondyle
		SENSORY	Little finger metacarpo-phalangeal joint	3-4 cm distally over DIP joint	Medial wrist adjacent to FCU tendon

LOWER LIMB	PERONEAL NERVE	MOTOR	Dorsal lateral foot over Extensor digitorum brevis muscle belly	Metatarso-phalangeal joint of little toe	1.Ankle anteriorly slightly lateral to tibialis anterior tendon 2. Head of fibula
	TIBIAL NERVE	MOTOR	1 cm proximal and 1 cm inferior to navicular prominence over Abductor hallucis brevis muscle belly	Metatarso-phalangeal joint of great toe	1.Medial ankle- slightly proximal and posterior to medial malleolus 2. Popliteal fossa- Mid-posterior knee over popliteal pulse
	SURAL NERVE	SENSORY	Posterior ankle posterior to lateral malleolus	3-4 cm distally at posterior ankle	Posterior lateral calf
PHRENIC NERVE		MOTOR	Two fingerbreadths (5cm) above xiphoid process over diaphragm muscle	Anterior costal margin 16 cm from G1	1.Lateral neck posterior to sternocleidomastoid muscle, approximately 3 cm above the clavicle 2. Anterior lateral neck between sterna and clavicular heads of SCM, just above the clavicle

NORMAL VALUES

	NERVE	RECORD	AMPLITUDE	CONDUCTION VELOCITY	ONSET (FOR MOTOR)/PEAK (FOR SENSORY) LATENCY	MINIMAL F-WAVE LATENCY
UPPER LIMB	Median nerve	Abductor policis brevis	>5.0 mV	>45 m/s	<4.3 ms	<31.2 ms
		Sensory	>11 microV	≥50 m/s	<3.6 ms	
	Ulnar nerve	Abductor digiti minimi	>5.5 mV	>43 m/s	<3.5 ms	<32.1 ms
		Sensory	>11 microV	≥50 m/s	<3.0 ms	
LOWER LIMB	Peroneal nerve	Extensor digitorum brevis	>2.0 mV	≥37 m/s	<5.4 ms	<59.3 ms
	Tibial nerve	Abductor hallucis brevis	>3.5 mV	>35 m/s	<6.1 ms	<54 ms
	Sural nerve	Sensory	>9.0 microV	≥40 m/s	<3.1 ms	
Phrenic nerve		Diaphragm	597 ± 139 (>320) microV		6.3 ± 0.8 (<8.0) ms	

F-WAVE PERSISTENCE-

MEDIAN NERVE- >50 %

ULNAR NERVE- >50 %

PERONEAL NERVE- >30 %

TIBIAL NERVE- >50 %

Any deviation from these normal values will be studied and compiles and results will be charted. These patients will be followed up till their stay in hospital for development of any type of respiratory involvement and then correlation will be made for any abnormalities in Phrenic Nerve conduction studies done during admission and results will be then charted..

7.1 Source of Data

Patients from DMCH Ludhiana admitted in Department of Medicine and Department of Neurology of DMCH Ludhiana with clinical suspicion of LGBS will be included in study from 1st November 2018 to 30th April 2020 and NCV studies including Phrenic Nerve will be performed on them and patients will be followed up till their stay in hospital.

7.2 METHOD OF COLLECTION OF DATA

All patients admitted to DMCH Ludhiana with suspicion of having LGBS. Then NCV studies including Phrenic Nerve will be performed on such patients to make the diagnosis of LGBS. Patients will be followed up till their hospital stay for any respiratory abnormalities and then these will be correlated with any Phrenic Nerve electrophysiological changes observed during NCV done during admission.

Phrenic nerve conduction studies are done with patient being supine with head slightly elevated and rotated to the side opposite to the nerve under stimulation. The phrenic nerve was stimulated percutaneously in the neck at the posterior border of sternocleidomastoid at the level of upper margin of thyroid cartilage. Then surface recording electrodes were placed in 8th/ 9th intercostals space with anterior electrodes in anterior axillary line.

All patients fulfilling the inclusion criteria will be included in the study and patients falling in exclusion criteria will be excluded.

INCLUSION CRITERIA

1. AGE ≥ 18 YEARS
2. Clinically suspected cases of LGBS
3. Diagnosis made on the basis of non-invasive electrodiagnostic studies i.e, NCV studies.

EXCLUSION CRITERIA

1. Other predisposing factors of polyneuropathy:
   i) Known case of Diabetes Mellitus
   ii) Known case of Hypothyroidism

NUMBER OF PATIENTS

All patients giving consent for enrolling in our study admitted to DMCH Ludhiana in Department of Medicine and Department of Neurology from 1st November 2018 to 30th April 2020, fulfilling the inclusion criteria but not falling under exclusion criteria will be considered for our study.

OUTCOME

Various abnormalities in Phrenic Nerve conduction studies will be observed in LGBS diagnosed cases and their role in giving an insight into possibility of patient developing some respiratory distress will be studied.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney *U* test for independent samples for parametric and non-parametric data respectively.

For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Nerve Conduction Studies including Phrenic nerve conduction studies

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached along with

LIST OF REFERENCES

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INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....

am willing to participate in the study **"PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME "** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____

गवाह के हस्ताक्षर

नाम:

_____

रोगी के हस्ताक्षर/अंगूठे का निशान

नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫ਼ਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"PHRENIC NERVE CONDUCTION ABNORMALITIES IN LANDRY GUILLAIN BARRE SYNDROME " ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।
ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____
ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

_____
ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਮਿਤੀ_____

ਪਤਾ _____

ਪਤਾ _____

_____
ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____
ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ

PROFORMA

Sr. No. ___

Identification Data-

- Name — Age — Sex
- Occupation — CR No./MRD No.
- DOA — DOD
- Contact number
- Residential Address

Presenting complaints-

Past history-

- Any Comorbidity
    - Known case of diabetes mellitus
    - Known case of Hypothyroidism

General physical examination-

- Pulse
- BP
- Respiratory rate
- Single breath count

Systemic examination-

- Abdomen

Signature of Candidate **Signature of Supervisor**

- Respiratory System

- CVS

- CNS

    - Higher mental functions

POWER	RIGHT	LEFT
UPPER LIMB		
LOWER LIMB		

TONE	RIGHT	LEFT
UPPER LIMB		
LOWER LIMB		

Signature of Candidate

Signature of Supervisor

DEEP TENDON REFLEXES	RIGHT	LEFT
PLANTAR REFLEX		
ANKLE REFLEX		
KNEE REFLEX		
BICEPS REFLEX		
TRICEPS REFLEX		
BRACHIORADIALIS		

SENSORY SYSTEM:

CRANIAL NERVE DEFICITS:

Provisional diagnosis-

Laboratory investigations-

1. NCV impression

Signature of Candidate

Signature of Supervisor

Motor Nerve Conduction Study

Site	Onset Lat.	N.D.	Amp.	N.D.	Segment	Dist. (mm)	Intvl. (ms)	NCV (m/s)
Phrenic	Left				Temperature:			
Phrenic	Right				Temperature:			
Median	Left				Temperature:			
Wrist		< 4.2		> 5	*Wrist			
Elbow					Wrist-Elbow			
Median	Right				Temperature:			
Wrist		< 4.2		> 5	*Wrist			
Elbow					Wrist-Elbow			
Ulnar	Left				Temperature:			
Wrist		< 3.5		> 5.3	*Wrist			
Elbow					Wrist-Elbow			
Ulnar	Right				Temperature:			
Wrist		< 3.5		> 5.5	*Wrist			
Elbow					Wrist-Elbow			
Peroneal	Left				Temperature:			
Ankle		< 5.4		> 2	*Ankle			
Head of fibula					Ankle-Head of fibula			
Peroneal	Right				Temperature:			
Ankle		< 5.4		> 2	*Ankle			
Head of fibula					Ankle-Head of fibula			
Tibial	Left				Temperature:			
Ankle		< 6.1		> 3.5	*Ankle			
Popliteal					Ankle-Popliteal			
Tibial	Right				Temperature:			
Ankle		< 6.1		> 3.5	*Ankle			
Popliteal					Ankle-Popliteal			

Sensory Nerve Conduction Study

Site	Post. Lat.	N.D.	Amp.	N.D.	Segment	Dist. (mm)	NCV (m/s)	N.D.
Median	Left				Temperature:			
Wrist		< 3.6		> 11	Wrist			
Median	Right				Temperature:			
Wrist		< 3.6		> 11	Wrist			
Ulnar	Left				Temperature:			
Wrist		< 3		> 11	Wrist			
Ulnar	Right				Temperature:			
Wrist		< 3		> 11	Wrist			
Sural	Left				Temperature:			
Sural					Sural			
Sural	Right				Temperature:			
Sural					Sural			

Signature of Candidate

Signature of Supervi

F-wave

Nerve	Side	Stim.Site	F-Lat.	F-Lat.N.D.	M-Lat.	F-M Lat.	F-Occurr.
Median	Left	Wrist		< 31.2			0 /0 .0 %
Median	Right	Wrist		< 31.2			0 /0 .0 %
Ulnar	Left	Wrist		< 32.1			0 /0 .0 %
Ulnar	Right	Wrist		< 32.1			0 /0 .0 %
Peroneal	Left	Ankle		< 59.3			0 /0 .0 %
Peroneal	Right	Ankle		< 59.3			0 /0 .0 %
Tibial	Left	Ankle		< 54			0 /0 .0 %
Tibial	Right	Ankle		< 54			0 /0 .0 %

Signature of Candidate

Signature of Supervisor

NERVE	F-WAVE PERSISTENCE
MEDIAN NERVE	
ULNAR NERVE	
PERONEAL NERVE	
TIBIAL NERVE	

Final diagnosis-

Treatment Given-

Outcome-

A. Any respiratory compromise/Discharged/Death

Signature of Candidate

Signature of Supervisor